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SURGERY OF THE PROSTATE GLAND.[1]

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The subject of the surgery of the prostate gland is one that has vexed practitioners for generations, and is even now far from being in a satisfactory state. That advances have been made in treatment and many patients relieved of tortures worse than death itself; that operations once almost surely followed by death are now almost certain to succeed; that the profession is becoming more unanimous in regard to the indications for one operation rather than another in certain cases; and that an operation is no longer needlessly postponed until the patient has at least one foot in the grave—these are all signs of the ceaseless activity of the modern surgeon in searching unremittingly, conscientiously, and successfully for the greatest good obtainable for those unfortunates who come under his care suffering from the distressing effects of hypertrophy of the prostate gland.

That many patients will do well in a life of catheterism, while some will begin to fail immediately it is commenced, is a fact now well recognized, and it is the purpose of the present paper to urge an early resort to operative measures in those patients in the latter class, believing, as I do, that operation here, though necessarily an evil, is yet a less evil when undertaken early than when postponed until the last possible moment.

The rise of prostatic surgery has been well sketched by Gouley, and it is from his address that many of the following references have been obtained.

It appears that the ancients considered that patients with prostatic hypertrophy suffered from "excrescences" or "carnosities" at the

[1] Read before the College of Physicians of Philadelphia, April 6, 1904.

neck of the bladder, and that when these offered obstruction to the evacuation of the bladder, their destruction was attempted with metallic instruments, introduced, of course, through the penile urethra. The existence of the prostate gland was unknown until the middle of the sixteenth century, its discovery being attributed to Nicolaus Massa.

Riolanus was the first to suggest, in the early part of the sixteenth century, that the bladder could be obstructed by a swelling of the prostate. This surgeon in several cases of urinary retention successfully practised incision of the neck of the bladder through the perineum, but it is not recorded whether the cause was enlargement of the prostate gland.

John Hunter, Sir Everard Home, Brodie, and others, both recommended and practised tunnelling of the obstructing body by the catheter; but this remedy was finally abandoned as dangerous. Chopart records that when Astruc, ten years before his death, which occurred in 1766, was attacked by retention of urine, Lafaye attempted to introduce a catheter, but met with an obstruction from a tumor in the neck of the bladder. He therefore perforated this by a lance-shaped stylet introduced through the catheter, which was open at the end, and by this means succeeded in forcing the catheter into the bladder and drawing off the urine. The catheter was retained fifteen days. This false passage through the obstructing body persisted, and a catheter was introduced by it as occasion required through the remaining ten years of Astruc's life, and at the post-mortem examination the condition of the parts was found to be just as described. Chopart himself tried tunnelling of the prostate several times, but with fatal results.

Systematic compression, to maintain a patulous urethra, first proposed by Physick, of Philadelphia, was employed by Leroy d'Etiolles and Mercier; but probably its best known advocate was Mr. Reginald Harrison, of London, who employed olivary bougies, which were passed once in forty-eight hours or less often. The bougie was so shaped that the olivary swelling caused dilatation of the urethra and compression of the prostate as the instrument was passed into the bladder and again as it was withdrawn, it being allowed to remain in place for several minutes.

Perineal operations came into favor earlier than those by the suprapubic method, owing probably to the greater familiarity of surgeons with operations in the former region, due to the then widespread practice of perineal lithotomy. Covillard,[1] in 1639, successfully operated by perineal cystotomy and removed a hard mass, not a stone, crushing and destroying it during extraction with the forceps. Chopart describes how Desault, who died in 1795, after

[1] Sir Henry Thompson considered the "excrescence" a true tumor of the bladder, and not part of an hypertrophied prostate; but Gouley speaks as if it were prostatic.

removing a calculus by perineal lithotomy, found and twisted off a tumor in the bladder, and Sir William Blizzard several times before 1806 performed perineal prostatotomy for enlargement without any calculous formation. Such an operation, combined with lithotomy, was by no means infrequent in the early part of the nineteenth century, and was sanctioned by Sir William Ferguson, who employed this procedure even before 1848.

Amussat removed a calculous and a protruding mass of the prostate by suprapubic cystotomy before 1832.

But the first regular surgical procedure was established in 1834 by Guthrie, under the name of "division of the bar at the neck of the bladder," this bar being in some cases due to the enlargement of the two lateral lobes of the prostate drawing up a fold of mucous membrane across the vesical orifice of the urethra. He accomplished his purpose by a catheter carrying a concealed blade. Where marked prostatic enlargement coexisted he advised perineal prostatotomy, but it is not certain that he ever performed it. Mercier, whose name is pre-eminent in the early days of prostatic surgery, devised, in 1837, special instruments—called by Gouley prostatotome and prostatectome—and at later dates modified them in various ways. These instruments were used as the internal urethrotome is at the present time, through the penile urethra, without either a perineal or a suprapubic opening, and did not meet with very general favor. Leroy d'Etiolles, as well as Civiale, claimed priority over Mercier in the introduction of the operation since known by his name, but it appears that their claims are ill-founded.

A further improvement on Mercier's method was that introduced about 1873 by Bottini, who aimed to avoid the hemorrhage attendant upon Mercier's operation by the use of galvanocaustic apparatus. Gouley, however, states that the bleeding by Mercier's method was trifling, and that therefore Bottini's modification was unnecessary. Although there are occasional reports of cases treated by Bottini's method during the twenty years or more following his first description of it, yet they are by no means frequent until after the publication, in 1897, of the well-known paper by Freudenberg, in which he described many improvements in the requisite apparatus. This surgeon, four years later, recommended the addition of a centimetre scale to the shaft of the Bottini cautery, in order that the operator might have a more definite idea of the position of the beak of the instrument when in use. Further modifications of the galvanocaustic apparatus have been introduced by Dr. H. H. Young, of the Johns Hopkins University; he, with Willy Meyer, of New York, and Orville Horwitz, of Philadelphia, being among the most prominent advocates at the present time of this operation, to the practical exclusion of all others.

Meanwhile, various other methods of treatment had been introduced. Of these, the most important are those that arose from the

practice of tapping the bladder in cases of retention of urine where passage of the catheter proved impossible. Simple catheterization to relieve the bladder of its residual urine had long been employed; and it is interesting to note that the clever manœuvre of increasing the curve of the catheter by partially withdrawing the stylet as its beak approaches the obstruction was practised and taught by Physick, the Father of American Surgery, long before it was accidentally discovered by Mr. Hey. Where it was impossible to introduce the catheter the bladder was punctured, either suprapubically or through the rectum. It was feared that urinary extravasation in the former situation would be more dangerous, and hence the majority of surgeons preferred the rectal route, it being found in many instances that the channel thus formed served fairly well for voluntary micturition (even after the cannula was withdrawn) until the urethra again became patulous by the subsidence of inflammation. Perineal puncture, though practised during the seventeenth and eighteenth centuries, fell into disuse during the early part of the nineteenth, the rectal route being the favorite.

Thus arose the treatment by urinary fistula, and from the concomitant drainage of the bladder, it may be considered a distinct advance in therapeusis. Sir Henry Thompson narrates that he saw some patients of Mr. Thomas Paget, who had had their bladders punctured suprapubically, completely relieved of the tenesmus and other distressing features by wearing a cannula or a catheter in the suprapubic fistula, and that this sight gave him encouragement to try the effect of permanent suprapubic drainage, even in cases with no retention of urine. When a suitable case presented itself, he accordingly introduced a long curved sound through the urethra, and, making it impinge upon the wall of the bladder above the pubic symphysis, cut down upon it with a small incision, and introduced a tube. In many cases in which he employed this method the relief afforded was marked, but he later abandoned this plan of treatment for drainage through the perineum. Dittel, Keyes, and Swinford Edwards were among the other surgeons who recommended the suprapubic fistula.

An important improvement in the method of forming the suprapubic fistula was that introduced in 1888 by Hunter McGuire. This consists, in brief, in establishing a fistula through the abdominal walls obliquely upward from the bladder, so that the fistula "bears the same relation to the bladder that the spout of a coffeepot does to the bowl." By this procedure McGuire was able to completely relieve his patients of their cystitis and residual urine, no involuntary leakage occurring even in the supine position, and the patients, in some instances, being able to project the stream of urine in a parabolic curve to a distance of several feet by voluntary contraction of the bladder. The urine was retained for from two to six hours. Morris, of New York, in one instance clothed this fistulous

tract with skin by transferring narrow cutaneous flaps into the wound at the time of operation.

The treatment by perineal fistula developed as a natural consequence of perineal cystotomy, and was generally combined with an incision into the prostate gland. This was a method largely employed by Reginald Harrison, commencing in 1881, his first operation having been performed on November 4th of that year. He used a small perineal incision, opening the membranous urethra; then the prostate was incised, and a perineal tube was introduced and retained for from six to twelve weeks. If the natural channel was not restored the fistula persisted. Prof. Gouley, of New York, claimed priority over Harrison in the re-introduction of perineal prostatotomy, his first operation, in which, however, he left no instrument in the bladder, having been performed April 7, 1880, and his third operation, in which he left a large-sized rubber tube in the perineal wound, having been done in January, 1881. Whitehead was likewise among the earlier advocates of the treatment by a permanent perineal opening.

Excision of the obstructing parts of the enlarged prostate by suprapubic cystotomy was first widely advocated by McGill, of Leeds, in 1887, he having before this date practised permanent suprapubic drainage, which he preferred to that by the perineum. Belfield, in America, had done suprapubic prostatectomy before this time, his first operation being in October, 1886; Dittell had, in 1885, removed a portion of an obstructing prostate through a previously existing suprapubic fistula, which he enlarged for the purpose; Trendelenburg, in May, 1886, and Benno Schmidt, in August of the same year, had employed this route for removal of pieces of the prostate, but to McGill has always rightly been attributed the priority in bringing this procedure prominently before the profession. The most enthusiastic supporters of McGill's operation have been Buckstone Brown, Kümmel, Atkinson, Keyes, and Fuller. As originally practised, this operation consisted in cutting off, through the usual incision for suprapubic cystotomy, by means of scissors or rongeur forceps, twisting off with bladder forceps, strangulating with an écraseur, or crushing with a lithotrite, any projecting masses of prostate tissue. At times, also, portions of tissue, forming the so-called prostatic tumors, were enucleated with the finger, either alone or aided by the scissors or other instrument, from their position deep within the gland.

But to Freyer is undoubtedly due the credit of bringing before the medical world a plan of operation whereby an attempt is made to enucleate the whole of the enlarged prostate gland. Through a suprapubic incision he opens the bladder, and incises the mucous membrane overlying the affected gland by a cut parallel to the urethra, over each lateral lobe of the prostate. Then, by working merely with the finger, he states that he has been able to enucleate

the entire prostate, leaving the urethra intact. The technique of this operation will be discussed in greater detail on a subsequent page. Many other surgeons have assailed Mr. Freyer's claim to originality, and assert that he is laboring under a grave misapprehension if he thinks he is the first person to operate in this manner, insisting that his method is nothing more than the removal of very large prostatic tumors from the substance of the gland, leaving behind the outer margin of glandular substance which, by the growth of these tumors, has been compressed into a thin capsule-like layer. Thus, Wallace says: "The more rapidly growing areas (of the diseased prostate) increase at the expense of the more slowly growing ones, which are compressed and stretched over the surface of their quickly growing neighbors. By this process a capsule is formed, ill-defined at first, but later becoming very distinct. The elements forming this capsule show in process of time a lamellar disposition. The adenomatous mass can now be easily enucleated, and not only presents a smooth surface, but also leaves behind a smooth cavity." One prostate which he describes, removed post-mortem, showed within the layers of the "capsule" a small lenticular focus of glandular tissue. He concludes: "These facts seem to leave no reasonable doubt that the so-called total prostatectomy is nothing more than the removal of adenomatous masses."

It seems a pity that so many controversies in regard to surgical priority are so constantly arising, and it appears that prostatic surgery is particularly unfortunate in this respect. Riolanus bitterly denounced his contemporaries for claiming as their own operations which had been employed before their grandfathers were born and for a hundred years before that time. Mercier asserted that Leroy d'Etiolles has assumed not only the credit of operations which were not his own, but had also assumed a name not his own, since, in reality, Leroy came from Paris and not from Etiolles. Gouley spoke almost venomously against Mr. Harrison; and I think Mr. Freyer would be well able to reply to his critics as Mr. Harrison did to Prof. Gouley: "I see that Dr. Gouley claims priority for the proceeding just described; what is of more importance is that it has received his approval."

Prostatectomy by the perineal route, developing from perineal prostatotomy, has been supported by Dittell, Annandale, Harrison, Zückerkandl, Watson, Syms, Ferguson, Moore, Murphy, Bryson, and others.

The simplest perineal operation is done through a straight median incision. To gain more room, this has been supplemented by an oblique cut on each side, as advocated by Murphy, while Zückerkandl advises a transverse semicircular incision, with its concavity toward the rectum, which is separated from the anterior structures by blunt dissection, and Dittel aims to get still more room by an incision completely encircling the right side of the anus from the

coccyx and continued forward in the median line of the perineum; by this approach he is enabled to remove a wedge-shaped piece of each lateral lobe. The coccyx may be excised if necessary for completing the operation.[1]

Combined operations by the perineal and suprapubic routes have been advocated by Nicoll, who pushes the prostate into the perineum (whence it is removed) by the fingers of one hand introduced into the bladder through the suprapubic wound, and are employed by a number of operators at the present day, including Alexander and Guitéras, some of them removing the prostate through the perineum, and some through the suprapubic wound, using the counteropening to admit the fingers of the other hand to assist in the manipulations.

A mode of treatment by castration introduced in 1893 by J. William White, though widely employed by some surgeons for several years, is no longer in favor. Vasectomy, suggested as a less severe and mutilating operation by Mears, seems likely to continue in use for certain cases for a long time. The mortality from castration for enlarged prostate is at least 18 per cent., taking all cases together, and in selected cases has been reduced only to about 8 per cent. Castration for enlarged prostate was first performed by Ramm of Christiania, and has been advocated by Griffiths and Mansell Moullin.

Ligation of both internal iliac arteries, to induce ischæmic atrophy of the prostate, was proposed by Bier and employed by him in three cases, one of the patients, operated on intraperitoneally, dying from septic peritonitis. Willy Meyer employed this operation in two cases, the first patient recovering, after secondary hemorrhage and partial gangrene of the left foot, but the second died, apparently of renal disease. Those patients who survived (3 out of 5) are said to have had their bladder troubles more or less relieved.

ANATOMY. In the human fœtus the prostate gland is a paired organ consisting of two lateral masses which coalesce around the first part of the urethra about the fourth or fifth month of intrauterine life. In some of the lower animals these lateral portions remain distinct and never unite in the median line. These facts have been known for centuries, and it was not until about one hundred years ago that Sir Everard Home took it upon himself to name as a "third lobe" that portion of the prostate lying above the ejaculatory ducts and behind the urethra. His experiences were apparently confined to an examination of four or five cases, the majority of which did not show any distinct third lobe. It is further worthy of observation that almost every author since his time has spoken of the "so-called third or median lobe" of the prostate as if there were reasonable doubt whether such a thing really existed.

[1] Perineal prostatectomy for malignant growths was employed in 1866 by Küchler, about 1870 by Billroth, in 1873 by Demarquay, in 1876 by Langenbeck, in 1882 by Spanton, and in 1883 by Leisrink. See Leisrink, Archiv f. klin. Chir., 1883, Bd. xxviii. s. 578.

Projecting into the prostate gland from the urethra is found the "uterus masculinus," which is the homologue of the female womb, being the persistent lower extremity of the Müllerian duct. This structure has a muscular and glandular wall of its own, and though in such close relation with the prostate, has no physiological connection with it; that is to say, the prostate gland, though composed largely of unstriped muscular tissue, can in no respect be considered homologous with the uterus.

Microscopically the prostate is found to be a compound tubular gland, its numerous acini being supported in a framework of connective tissue and involuntary muscle. The involuntary muscle is arranged in a rather compact layer around the periphery, and also at the site of coalescence of the two lateral lobes, above and below the urethra. The unstriped muscular fibres of the bladder wall are prolonged around the prostatic urethra as an inner longitudinal and an outer circular layer.

The whole gland is enveloped by a fibrous capsule distinct from the rectovesical layer of the pelvic fascia and formed by a condensation of the stroma of the gland. Outside of this fibrous capsule, and between it and the pelvic fascia, is found the prostatic plexus of veins, together with the arteries, lymphatics, and nerves distributed to the glandular structure. The venous plexus lies chiefly on the anterior and front lateral portion of the gland, receiving the dorsal vein of the penis.

Above, the prostate is in intimate relation with the urinary bladder, lying immediately beneath its neck, and being separated from its muscular walls only by a layer of fascia; below and posteriorly it impinges upon the rectum, connected rather firmly to this structure by a prolongation of the rectovesical fascia. In some instances the prostate gland has been found to envelope only about three-fourths of the calibre of the urethra, this canal merely grooving the gland upon its upper and anterior surface; but in the vast majority of cases the urethra appears to be channelled out of the substance of the gland, somewhat anterior to its mid-portion.

Besides the support it receives from the bladder and rectum the prostate is suspended in the anterior fibres of the levator ani muscle, called by Santorini the levator prostatæ, and anteriorly is supported by the puboprostatic ligaments, or true ligaments of the bladder (prolongations of the rectovesical fascia); while in the median line anteriorly, where the rectovesical fascia and the deep layer of the triangular ligament unite, the prostate is in relation with this latter structure, a levator prostatæ muscle being on each side.

Coursing through the posterior mid-septum, dividing the two lateral lobes, are found the ejaculatory ducts of the seminal vesicles and vasa deferentia. They empty into the floor of the prostatic urethra on each side of the caput gallinaginis, and posterior to the orifice of the uterus masculinus. The prostatic acini of each lobe

likewise empty into the prostatic urethra by eighteen or twenty ducts on their respective sides of the median line.

Where a projection into the bladder posterior to the vesical orifice of the urethra appears to arise from the prostate, it has been said (Griffiths) in some instances to spring from an accessory prostatic gland, which might then well be called a third lobe. But in the majority of cases such a growth may be and frequently has been traced to one or other of the two lateral lobes, being connected to them by a pedicle, much as a subperitoneal fibroid is attached to the uterus. It seems, indeed, not impossible that in those cases where it has been said that no such attachment could be found, the growth had finally torn its pedicle loose, and might in time even have migrated, as is not infrequently the case with analogous tumors of the uterus. Be these facts as they may, it is undoubtedly true that most surgeons at the present day are inclined to regard the third lobe as non-existent, that portion of the gland formerly called the third lobe, and lying between the two lateral lobes, above and posterior to the ejaculatory ducts, being recognized as merely the area of coalescence of these lateral lobes.

ETIOLOGY. The causes of enlargement of the prostate are still involved in much obscurity. Our ideas appear to have become less positive in this respect in proportion to the increase of our knowledge in other directions.

In the first place, nevertheless, practically everyone is agreed that there are two main pathological processes by which enlargement of the prostate gland is effected:[1] one by means of a tumor formation—a fibromyomatous, fibroadenomatous, or adenomyomatous change—and the second by a general increase in the size of the organ without distinct tumor formation. Both processes may coexist.

The tumors may be several in number, or there may be only one; they may arise in the substance of the gland or beneath its capsule. They may be composed largely of glandular structure, of muscular tissue, or of fibrous tissue; probably a combination of the first two is the most frequent, producing adenomyomata; the second in frequency of occurrence being the fibroadenomata, for in scarcely any instance is there any adenomatous tissue whatever.

Where general increase in size occurs, approaching more nearly a true hypertrophy, the fibrous is usually the predominant tissue, and it is this type of enlargement which is, as a rule, the cause of the stony hardness observed in some prostates.

To ascertain the underlying causes of the enlargement, whether neoplastic or hypertrophic, affecting the prostate, a number of theories have been proposed. These may all be reduced to two classes, in one of which the enlargement is held to be primary—to

[1] Griffiths thought that the adenomatous change was merely the first stage, of which fibrous overgrowth formed the final change, Journal of Anatomy and Physiology, 1889, vol. xxiv. p. 236.

be itself a cause—to arise *de novo;* and in the other of which it is considered a secondary change—as the result of some other process —as, perhaps, a misdirected compensatory hypertrophy.

To be strictly sincere, it does not appear to me that it is a matter of very great importance under which of these two theories we enlist our intellect; for it is, after all, a matter of purely academic interest, and for practical surgeons will remain such until some happy discovery will reveal the true cause of tumor formation in general, or the reason why at a certain age the bodily powers begin to fail and the tissues to become sclerotic.

To my mind those prostates which show tumor formation are to be classed with other tumors, which were formerly defined as discontinuous, purposeless hypertrophies of no known cause; while those glands which show only general hypertrophy are classed with other changes incident to age. The discussion of the causes has, I think, been much confused by the neglect of writers to keep these two classes distinct. Guyon and his school, who held that such enlargement was merely a part of a general senile change, appear to me to confine their considerations to the latter class; and here, too, may be placed the theory of Harrison, that the hypertrophy results from a vain effort to empty a bladder whose base has previously descended into the pelvis below the level of the urethra.[1] While, on the other hand, Velpeau, Paget, and Billroth, with their followers, who maintained that these phenomena were due to a myomatous change strictly analogous to that found in the uterus, evidently fixed their attention exclusively on cases of what I have called the first class, which is, in fact the larger.

In discussing the clinical causes, also, it is well-nigh impossible to avoid theorizing, since, as I have said above, so little is accurately known of the causes of this affection.

Race does not exert a very marked influence; the negro race is probably slightly less predisposed than is the white (Conner), and among the natives of China and Japan prostatic troubles are considered to be extremely rare. In India and Turkey, however, they are comparatively frequent (Wishard).

Age appears to exert a very marked influence. It is rare for any symptoms to be produced by an enlarged prostate before a patient has reached the age of fifty years; but Dr. Mudd has seen cases in a young negro, aged twenty-seven years, in a child, aged five years, and in an infant, aged thirteen months. The researches of Thompson and others have shown that appreciable enlargement exists in about one-third of persons over sixty years of age, but that it produces manifest symptoms in only about one in every twenty. For prostatic enlargement to appear first after the seventieth year is very

[1] Were this theory correct, a cure might be hoped for, in cases not too far advanced by a ventrosuspension of the bladder.

unusual. In natives of India and other tropical climates it appears much earlier, as a rule, the span of life being about fifteen or twenty years shorter than ours.

Social habits exert some influence. The gouty, those who have been addicted to the excessive use of alcoholic beverages, or who have been high livers in other respects are believed to form a large proportion of these patients. Why this should be so; why such a life should produce enlargement of the prostate gland, has not been determined. Excessive sexual indulgence has been held responsible in some instances, the overexercise of the gland being thought to lead to hypertrophy, just as Harrison thought hypertrophy ensued from constantly recurring efforts to empty an atonic bladder. This "prostatic overstrain," so named by Lydston, has been explained in the following way by Harrison: "That the withdrawal of a portion of that function of the prostate in which it has been the most vigorously engaged, should be followed by a continued activity in which growth is substituted for secretion, is not, I consider, pathologically illogical." McGuire held that, although prostatic overstrain might be the true cause of the enlargement, yet it was the senile sclerotic changes in the bladder which first caused the enlarged prostate to give symptoms.

Preceding inflammations, such as gonorrhœa, acute or chronic prostatitis, or prostatic abscess, may also be the starting-point of chronic enlargement of this organ.

Chronic passive congestions, finally, as in other organs, may lead to prostatic enlargement as well. Old men with cirrhotic kidneys and liver, with hemorrhoids or with varicose veins in the legs, are very apt to be sufferers from this malady.

Clinical Pathology. As the prostate gland enlarges, whether from tumor formation or as the result of a general hyperplastic process, various changes are produced in the bladder, the urethra, and the rectum. The usual direction of greatest enlargement is parallel to the urethra and posteriorly; that is, in the direction of least resistance, toward the base of the prostate, and away from the triangular ligament, this latter attachment of the prostate being practically a fixed point. The bladder cavity is likewise usually encroached upon, the vesical mucous membrane being raised posterior to the orifice of the urethra, and a pouch forming behind this projection. If the two lateral lobes enlarge in nearly equal degree, a mere fold of mucous membrane may be raised across the neck of the bladder just behind the vesical opening of the urethra, but some little distance in front of the ureteral openings. This fold constitutes the most usual variety of "bar at the neck of the bladder."

As the lateral lobes enlarge the prostatic urethra becomes lengthened, sometimes measuring as much as four inches, instead of barely an inch, which is the normal; and the curve is also much increased from the height to which the inner opening of the urethra is raised.

If one lobe enlarges more than the other the urethra is correspondingly deflected toward the less enlarged side. Where a submucous tumor projects into the floor of the bladder, at or near the median line, the urethral channel may become Y-shaped, and the catheter will pass now to the right, now to the left of the projecting mass. Finally, where the anterior commissure, or isthmus of the gland, enlarges as well,[1] there may be a collar formed around the vesical orifice of the urethra, which may then appear as a dimple, deeply seated near the centre of the prostatic mass.

At times where no intravesical growth at all is found, or after any such has been removed, it has been found still impossible to introduce a catheter through the urethra, the obstruction then being due to irregular projections into the floor of the prostatic urethra. This circumstance has probably been the cause of many failures to restore voluntary micturition after the old operation of McGill.

The calibre of the prostatic urethra sometimes becomes so much increased from the irregular enlargement of the prostate that it may contain an ounce or two of urine, and the unwary may be deceived into thinking the bladder has been reached by a catheter, which merely empties this little collection of urine in front of the real obstruction. On the other hand, the whole prostatic urethra may be compressed into a slit-like channel, impervious to fluid except when distended by an instrument. The vesical orifice may assume a crescentic shape, the concavity of the crescent being directed toward the area of greatest enlargement.

If the enlargement proceed chiefly toward the rectum that channel may be obstructed, and much difficulty in defecation ensue; while hemorrhoids may arise either consecutively or from a common cause.

Ordinarily, the first consequence of the almost invariable post-prostatic pouch formation is the presence of residual urine—or that urine which the patient is unable to expel by his voluntary efforts. Unless the prostatic enlargement is arrested, the amount of residual urine gradually increases, owing both to the increasing obstruction and to the decreasing muscular tone of the bladder, which becomes weakened by straining and overdistention. Ammoniacal decomposition of the urine, with consequent cystitis, quickly supervenes and gives rise to what is commonly the first symptom, namely, increased frequency of micturition.

As the residual urine accumulates the vesical orifice of the ureters may first become compressed, preventing the periodical discharge of their contents into the bladder and causing circulatory disturbances in the kidneys; while at a later period they may become dilated, along with the stretching of the rest of the bladder wall, so

[1] Dr. Murphy denies that this portion of the gland is subject to enlargement. Journal of the American Medical Association, March 29, 1902.

that they are no longer protected by a valve, but are in direct communication with the bladder, the backward fluid pressure reaching even the renal tubules. Finally, when the bladder becomes overdistended, a condition of urinary overflow may occur, and the patient will be forced to pass a drachm or less of urine every ten or fifteen minutes, or will suffer from a constant dribbling throughout the twenty-four hours. The combined backward pressure and septic changes in the urine produce renal disease, and this, in turn, reacts on the composition and quantity of urine.

The bladder may not, however, dilate; its walls may become much thickened, corrugated, and pouched; its cavity may even contract, or may be nearly completely filled by an adenomatous prostate. But, as a rule, where an adenomatous change occurs in the prostate the bladder dilates; whereas, if the prostate be a hard fibrous mass, and comparatively small, the vesical walls are contracted and thickened.

The weight of the diseased prostate varies from one to twelve ounces; yet the largest in bulk may not weigh as much as one smaller but more compact.

Uric acid, or phosphatic calculi, frequently form in the puddle of residual urine, and much increase the suffering of the patient. Hemorrhage may thus be accounted for.

Symptoms. The symptoms of prostatic enlargement may be conveniently considered under the heads of those of which the patient complains and those which the surgeon discovers on examination.

The patient will probably for a rather indefinite time have been affected, perhaps without paying much attention to it, with difficulty in starting his stream of urine; the urine when started will have been expelled with little or no force, tending to drop vertically from the meatus, not spurting forth in the normal parabolic curve; a longer time than usual will have been occupied in emptying the bladder, and at the conclusion of the act of micturition there will have been considerable dribbling of urine, no power existing to void the last drops in spurts. The stream will probably not be smaller than in health unless stricture exists as well.

But probably the first deviation from normal which will have forcibly arrested the attention of the patient will have been the necessity of arising once or oftener during the night to evacuate his bladder. This increased frequency of micturition may also have annoyed him during the day, but is less noticeable then that at night. The bladder may become so extremely irritable that no sooner will any urine have been discharged into it from the kidneys than an uncontrollable desire to empty the bladder will arise, and thus the patient will be reduced to a most miserable state, being impelled to urinate every ten or fifteen minutes, and his general health in a very short time showing the effects of this constant unrest. On the other hand, the residual urine may gradually accumulate without causing such

severe distress, and a state of retention with overflow finally develop. Besides the cystitis thus manifested by crebruria, a feeling of weight and distention in the perineum may exist, and piles, with prolapsus ani, from the consequent tenesmus, may ensue.

The patient may consult a physician before this stage is reached, or he may not be disturbed sufficiently to think this necessary until some exposure to cold, some indiscretion in diet, or some other irregularity in his life has brought on an attack of acute retention of urine. Thus, when first seen by the physician he may present a bladder distended nearly to the umbilicus, he himself not being aware that residual urine had been accumulating for perhaps months past. Hence, the first physical sign to be looked for in patients suspected of prostatic troubles is a hypogastric tumor, with the characteristics of a distended bladder. It is especially important to detect this before introducing a catheter in cases where chronic retention has probably existed for a long time; for, as is well known, the complete sudden evacuation of such a bladder is, as a rule, speedily followed by the patient's death.

The patient should next be requested to urinate, when much information may be derived from the character of the stream, in accordance with the changes noted above. If it is apparent that no excessive amount of residual urine exists, it will now be advisable to introduce a catheter, noting the presence of strictures, the distance of the vesical orifice of the urethra from the meatus, and any changes in the curve or deviations from the right line of the subpubic urethra. Then the amount of residual urine is determined, not being deceived by the ounce or more which may, in rare instances, flow from the dilated prostatic urethra. While the catheter or a sound is still in the bladder a finger should be introduced into the rectum, when, by careful manipulation of the one and palpation with the other, a fairly accurate idea can be obtained as to the size, form, and density of the prostate. The condition of the bladder walls, whether rugous and contracted, coated with phosphates, thickened, or dilated and overstretched, can also be ascertained, as well as the existence of a calculus.

If an obstruction to the catheter be encountered within less than seven inches from the meatus, it is in all probability due to a stricture and not to the prostate; but if the obstruction be easily overcome by sharply depressing the handle of the instrument, if of metal, or by slightly withdrawing the stylet, if of webbing, the obstacle is almost certainly an enlarged prostate.

Diagnosis. The symptoms and physical signs being well known, a diagnosis is not, as a rule, difficult. The most important affections with which enlargement of the prostate may be confounded are: atony of the bladder; cystitis, especially chronic; stricture of the urethra; vesical calculus; tumor or tuberculosis of the bladder, and malignant disease of the prostate or some neighboring structure in

the pelvis. By symptoms alone no one of these can be definitely excluded, but by careful attention to the clinical history, combined with painstaking physical examination, I believe that in almost every case a correct diagnosis can be reached.

In atony of the bladder arising from other causes the existence of an enlarged prostate may be readily excluded by combined exploration with an intravesical sound and a finger in the rectum, while in uncomplicated cystitis these same marks of difference hold good, and there is, moreover, no residual urine.

In stricture of the urethra the obstruction, as stated above, is almost invariably within seven inches of the urinary meatus, and an enlarged prostate may be excluded by the means of examination already indicated. In case, however, of an impermeable stricture with chronic retention it will not be possible to satisfactorily examine the prostate until these conditions are relieved.

Where a vesical calculus exists it is not liable to be mistaken for an enlarged prostate unless it is both encysted, and so thickly coated with mucus that no grating sensation is imparted to the sound. But even under such circumstances there may be no residual urine, which is, as already insisted upon, a nearly invariable accompaniment of every enlarged prostate producing symptoms, and there will probably not be the characteristic change in curve of the subpubic urethra. If the calculus be prostatic, or even if it merely coexist with an enlarged prostate, a positive diagnosis is more difficult. Bleeding is more common in cases of calculûs than in those of enlarged prostate alone, and the pain is less constant and more confined to times when the bladder contracts upon the concretion or when the patient is actively moving about. The greatest frequency of urination, also, is during the day, and the patients with stone are not, as a rule, disturbed much at night.

Probably the most difficult diagnosis of all is that from polypoid growths in the bladder, which, when springing from the region of the prostate, may very closely simulate a pedunculated tumor of this organ. But in nearly all other forms of vesical tumor other than prostatic, hemorrhage is an early and conspicuous symptom, and is usually not attended by much pain.

Tubercle of the bladder is probably always secondary to similar disease elsewhere in the body, most often in the epididymis. While the vesical tenesmus may simulate that present in cases of enlarged prostate, physical examination with sound, finger, and cystoscope, and careful examination for tubercle bacilli and the application of the tuberculin test will usually reveal the true state of affairs.

Malignant growths of the prostate itself can be distinguished from benign enlargement of this organ only by the tendency they exhibit to infiltration if of carcinomatous origin, or to rapid growth and the production of cachexia if of a sarcomatous nature. Compared to the benign overgrowths they are distinctly rare.

PROGNOSIS. A question of considerable importance and much interest in connection with enlargement of the prostate is that of prognosis. In few other diseases is it so necessary for the surgeon to know what may be accomplished by the various methods of treatment possible, and in probably no other class of cases is the surgeon blamed more severely for errors in judgment. It is not sufficient, indeed it is neither ethical nor humane, to hope that the patient will die of some intercurrent affection before any necessity arises for instituting active treatment on behalf of his enlarged prostate; and hence every physician or surgeon who has such cases under his care must give careful thought and attention to each individual patient, and must know whether the expectation of life will be lengthened or decreased by the treatment he purposes undertaking, or whether the certainty of a life of considerable discomfort for a longer time is not less to the patient's ultimate advantage than the risk to life incurred by a rather severe and shocking operation, which, if successful, will enable the patient to live out his natural term of life in ease and comfort.

There are, then, two main questions to be solved in this connection; first, whether the patient's life can be saved, prolonged, or, at least, not sacrificed, by the treatment to be pursued; and second, whether his sufferings will be relieved wholly or in part, or whether no change at all can be obtained.

Under medical treatment and catheterism there is practically no possibility of directly terminating the patient's life; with the understanding that every antiseptic precaution be taken in catheterization, his life may be prolonged, and, in certain cases, made very comfortable.

Many a patient who has to pass a catheter only once or twice in the twenty-four hours will live a life of perfect ease, and will round out his days without interruption. But, where the catheter has to be passed frequently, that is to say, as often as four to six times in the twenty-four hours, or where its passage at even longer intervals is attended with pain or difficulty, catheterism must be considered at the present day an insufficient remedy, except in those who are already on the threshold of the grave. The expectation of life, moreover, in patients treated by catheterization has been shown by Harrison and Lydston to be, in the average, not more than four or five years.

The next mildest form of treatment is drainage of the bladder. By this means may be obtained relief of the cystitis, and, consequently, of the tenesmus, pain, and general unrest, in a certain number of cases. In my opinion, it is applicable chiefly to those in a very debilitated condition or to the very old. Drainage by a permanent catheter introduced through the urethra can rarely long be endured, and is usually only to be employed in preparing the bladder for radical operation.

Cabot,[1] in an article on the effect of drainage on the kidneys in nephritis, makes some observations of very great interest in connection with the treatment of prostatics. Starting with the proposition that urinary obstruction from enlarged prostate or other cause produces increased renal pressure, which may even reach to the Malpighian glomerules, and that this pressure alone, even without infection, will result in fibrous changes (interstitial) in the kidneys, as well as in an increase in quantity and a decrease in specific gravity of the urine excreted, he goes on to show by the report of six cases that the invariable result of relief of the obstruction was to bring the urine back to a more nearly normal state. Especially noticeable was this result in cases where even the greatly increased secretion of urine had been ineffectual in "carrying off the waste products in sufficient amount" and where uræmic symptoms had appeared.

He is well aware, he states, that this procedure is no new therapeutic measure, but he thinks its applicability to renal disease has not hitherto been sufficiently appreciated, although the good effects of Edebohls' operation in relieving renal tension are well known to the profession. Those authors as Langlois, he adds, who have noted the decrease in size of enlarged kidneys succeeding upon bladder drainage, have thought it was due to either relief of congestion or to the emptying of hydronephritic pouches.

Dr. Cabot says that for the purpose of the present investigation he has "taken it for granted that a patient with obstructive disease of the prostate and dilated bladder may be regarded as having some degree of interstitial nephritis if the urine is abundant and of very low specific gravity. If such a patient is suffering from anorexia, nausea, and dry mouth, either with or without psychical disturbances, these are to be looked upon as symptoms of uræmia due to this interstitial nephritis. If, in addition to these symptoms, he has a high fever and the urine contains pus, it is to be regarded as probable that he has pyelonephritis." By instituting continuous drainage and by flushing out the urinary tract with large draughts of water, he has succeeded in promptly relieving such symptoms.

The time during which this constant drainage must be continued varies with the individual patient; by discontinuing it soo soon, the renal and uræmic symptoms were found to promptly recur, but by persisting in this treatment until the urine was nearly normal in both quantity and quality, his patients returned to a life of intermittent catheterization (when they declined operative interference) in the enjoyment of excellent health.

Dr. Cabot further insists that the dangers of sudden emptying of a chronically distended bladder are much mitigated by the constant drainage instituted; for he has found that although considerable hæmaturia might ensue for a few days, yet that this neither in any

[1] Boston Medical and Surgical Journal, 1903, ii., p. 559.

way interfered with the perfect drainage, nor was it followed by increasing uræmic symptoms, as is usually the case when such a bladder is emptied and no constant drainage established.

The first case reported is that of a man, aged sixty-seven years, who, after using the catheter for some months, there being over twelve ounces of residual urine, was taken severely ill, with a temperature of 103° F.—apparently commencing pyelonephritis. By constant drainage the amount of urine was, in the course of some weeks, reduced from 60 or 70 ounces to less than 50 ounces daily, while its specific gravity increased from 1007 to 1023. Intermittent catheterization was then instituted.

In the second patient there was anorexia, dry mouth, much nausea, and marked somnolence. By constant drainage the return to normal was remarkably rapid.

The third patient, in much the same general state, had fever as well; his bladder also was distended nearly to the umbilicus. Thirty-seven ounces of urine of very low specific gravity were withdrawn, and by continuous drainage, in a period of two months, the patient's health was practically restored, the quantity of urine, however, remaining at 70 to 90 ounces daily, and its specific gravity being about 1008.

The fourth patient, aged seventy-two years, had suffered for several years with crebruria, there being about 12 ounces of residual urine. A resort to catheterization once or twice daily produced, in about two weeks' time, symptoms of commencing pyelonephritis. Constant drainage was then begun. Hæmaturia occurred, and the next day the patient became uræmic. Under proper constitutional treatment all unfavorable symptoms disappeared, but it was not until the seventy-ninth day that intermittent catheterization could be substituted for continuous drainage. His daily urine remained at about 100 ounces, of specific gravity 1012 to 1015.

In the fifth patient the chronically obstructed bladder contained 40 ounces of urine. Constant drainage, while it produced transient hæmaturia, relieved the delirium, fever, and thirst. Three weeks later intermittent catheterization was begun. The urine at last report, over a year after the acute attack noted above, was 40 to 60 ounces daily in quantity, of specific gravity 1017.

The last patient reported was in the early stages of chronic nephritis, with polyuria amounting to 8 pints in the twenty-four hours. When chronic retention set in this amount was reduced to 6 pints, of specific gravity 1005. Constant drainage was instituted, and in one day the amount of urine drained off reached 16 pints. This was gradually reduced to 6 pints, and, finally, to 4 pints daily, while the specific gravity rose to 1012 and 1015.

The success of Thompson, McGuire, and others in treating these patients years ago by means of suprapubic permanent drainage, and of Harrison by means of a perineal tube, should not be forgotten

at the present day; and, while we recognize the inadequacy of such methods to restore the patient to his normal condition, yet, in a limited number of cases, they are still useful. Especially is this so in patients with very bad cystitis and where some immediate relief is imperative. In such cases so radical an operation as prostatectomy will almost surely kill, unless time can be obtained to relieve the cystitis, to get the kidneys into fair condition and to improve the general health of the patient. In patients such as these the formation of a permanent suprapubic fistula by McGuire's method, or, if the prostate be not too large, simple perineal prostatotomy with the introduction of a tube will afford almost certain relief to the urgent symptoms, and in many instances will enable the constitution to withstand prostatectomy at a later date. Neither castration, vasectomy, nor ligation of the internal iliacs will act sufficiently quickly in such cases; indeed, in my opinion, these operations are no longer to be considered desirable under any circumstances.

References which I have to 107 cases treated by McGuire's method show only 2 deaths; so that even in the most serious cases it will be exceedingly unlikely to precipitate a fatal termination.

The Bottini operation is not one for the general surgeon, nor, to my mind, for the average patient. On the surgeon's part it requires a technical knowledge, which, even in the hands of those known to be the most skilful in its employment, has not sufficed to prevent fatal complications. The greatest points in favor of galvanocaustic prostatotomy are its low mortality (4 to 6 per cent.), and the fact that if it fails to relieve after the first application it may be repeated once or several times with little more risk than attended its first employment. Moreover, the patient has still a prostate left whose removal by excision may effectually cure him, even when several applications of the Bottini cautery have proved ineffectual. On the patient's part, this operation is often welcomed for these very reasons: that its danger is comparatively slight, that general anæsthesia is not required, that no "cutting" is to be done, that no external wound is left for healing, and that it does not interfere with subsequent operative treatment by other methods.

But with all the advantages claimed for it, Bottini's operation is first and foremost a makeshift, quite as much as castration or McGuire's suprapubic fistula. It does not remove the offending organ. It burns grooves in it, and the surgeon leaves sloughs to be cast off into the bladder, and then waits, hoping that the cicatrization of his sloughing sores will diminish rather than increase the deformity. It is an operation done practically without the aid of either sight or touch; it is nearly impossible to know how long, how wide, or how deep are the grooves burned, or whether they are being burned in the prostate or in the bladder wall; and it is absolutely impossible to know what has been accomplished in the destruction of prostatic tissue or that the prostate will not again grow into an

obstructing position. Moreover, it is an operation wholly inapplicable to emergency cases and to patients with urethras impassable from either strictures or prostatic obstruction. It is, therefore, in my opinion, an operation only to be employed when (1) prostatectomy is inadvisable or is refused, or when (2) the urethra is freely open to instrumentation, there is no cystitis, and little residual urine.

We are reduced, then, to prostatectomy as the operation of first choice in all cases in which it is not contraindicated. When done by the method of Freyer, that is, by suprapubic enucleation with the finger, passing between the capsule and the sheath, the mortality is decidedly lower than by the method of McGill. In 12 cases of the former operation, Mr. Moynihan had only 1 death, and in 51 cases Mr. Freyer had 5; Barling, however reports 10 cases so treated, with 3 deaths, making, with 6 successful operations recorded by McRae and by Stoker, a combined mortality rate of less than 12 per cent. for the 79 patients; while the statistics of Belfield and of Mansell Moullin show, respectively, nearly 14 per cent., and over 20 per cent. for McGill's operation. Recent cases of McGill's operation, reported by Armstrong, Horwitz, Fuller, and Thorndike, show a death rate of over 21 per cent. among 28 cases. The mortality in 73 recent cases (Albarran, Deaver, Ferguson, Horwitz, Murphy, Syms, Verhoogen) of perineal prostatectomy is only a little over 8 per cent., but the results have not, as a rule, been nearly so good as those achieved by Mr. Freyer's method. Forty-six out of fifty-one patients treated by this latter operation are reported by him as having their urinary function restored to its natural condition.

Mere figures, therefore, are decidedly in favor of Freyer's operation; but both from what I have read in the reported cases of others and from my own experience, I am convinced that it is not the best operation in every case, even if it be possible. Mr. Freyer reports a case in which he attempted his own method, but owing to the density of the gland and its firm adhesions to the rectovesical fascia, he was able to remove only pieces of it. This patient died on the second day after operation of heart failure, and the bladder was found full of clots, the hemorrhage during the operation having been free. Now, it does not appear to me to be strictly sincere to argue that Freyer's operation gives a mortality of less than 10 per cent. when we exclude cases in which it was attempted but found impossible of performance. As well might we give a certain mortality for the operation of ovariotomy, considering only those cases where the adhesions to the intestines were few and light, and where the cyst shelled out easily, yet exclude those cases where a prolonged and delicate dissection was necessary to deliver the cyst, claiming that such cases form a class of themselves. Such a method of calculation would only lead others astray and encourage us in self-deception, a thing Mr. Freyer himself would be very far from countenancing.

Certainly, everything within my own experience convinces me

that while Freyer's method of prostatectomy is by far the best operation for all cases to which it is applicable, yet that there is a small and limited number of cases in which it is neither advisable, nor, as Mr. Freyer's own patient above referred to, plainly shows, at all possible.

The perineal operation I have employed a number of times, and have found it quite satisfactory in certain cases, but for some years have largely abandoned it for the operation of complete suprapubic extirpation, which I find in my own hands gives better functional results, is attended by a less mortality, and is certainly easier of performance. Of course, we know that mere facility of execution for the surgeon is no argument in favor of one operation, if another, even if much more tedious and intricate, would assure a better result to the patient; but the greater ease in the suprapubic operation consists, not alone in mechanical execution, but in economy of time and less distortion of the neighboring parts, both of which are considerations of importance in the class of patients under discussion.

TREATMENT. The treatment, therefore, of prostatics—a convenient term adapted by Belfield from the German *Prostatiker* and the French *prostatique*—resolves itself into a choice of remedies, not into any hard-and-fast rules which may not be transgressed. It has well been said that he is either a fool or at best a surgeon of very limited experience who knows of only one method of treatment for a certain class of cases; and while I myself maintain that prostatectomy by the suprapubic method of Freyer is the best treatment, I do so with the distinct reservation that it is not immediately applicable to every case.

Having seen, then, in the preceding pages the relative dangers and probabilities of ultimate success from the various methods of treatment applicable, it is next requisite to attempt an expression of some general rules which may guide the surgeon in selecting his course of treatment.

A healthy man somewhat under sixty years comes to you with the simple complaint that he finds it necessary to rise once almost every night to pass his water; that this is becoming, he thinks, a more invariable habit; that he has waited, thinking matters might return to their former state without treatment, but that he fears they are growing worse. In such a case as this, after finding the cause of his trouble to be a moderately enlarged prostate, and determining the amount of residual urine to be trifling—one or two ounces—I would recommend the passage of a soft rubber catheter, with strict asepsis, every night at bedtime. It is probable that the bladder being thus emptied, no call during the night would arouse him, and it is barely possible, though I admit very improbable, that with good hygienic surroundings and a regular life, the bladder might acquire sufficient tone to expel the residual urine, and that no further trouble would ensue. More likely is it that catheterization, at first employed only

once in the twenty-four hours, would soon, say in the course of six months to a year, be resorted to twice or even three times in a similar period, to obtain relief from the vesical tenesmus, and that from this constant use, cystitis, if only of a mild degree, would have developed. At this period of the disease it is important to undertake more active measures before the general health is impaired. In cases such as this very many surgeons would be disposed to recommend the Bottini operation—a trifling risk; no cutting, no external wounds to heal; a prostate not very large, nor causing much obstruction; possibly no impairment of the generative function as a result of the operation. The patient would be more inclined to accept such a proposition than one of radical extirpation of the gland, even though he were told that one operation by Bottini's method was not a sure cure, and that even after two such operations the prostate might still continue to enlarge. In my opinion, if the prostate gave evidence of rather rapid increase in size, showing it to be probably an adenomatous gland, a Bottini operation would not be for the best ultimate good of the patient; I would advise him to have a suprapubic prostatectomy done by Mr. Freyer's method. The prostate would probably continue to enlarge, even if temporary relief from the residual urine had been obtained by the galvanocaustic operation; and the patient would in time be forced to submit to a radical operation when periprostatitis had rendered complete extirpation impossible, when his bladder had become weakened, his kidneys diseased, and his general constitution impaired. If, on the other hand, the prostate in such a case appeared to be a small fibrous mass, not increasing in size, but obstructing the urine by pressure on the prostatic urethra, I should feel less disinclined to try the effects of a Bottini operation. This latter class of patients is distinctly rarer than the former. In the former case the prostate could almost certainly be removed in its capsule from within the sheath of the gland, with comparatively little hemorrhage and with practical certainty of no recurrence. In the latter case, however, the prostate would probably be densely adherent to its sheath; an attempt at extirpation by the suprapubic route would probably be difficult, bloody, and prolonged; the gland would have to be removed in pieces, not insuring its total ablation. but perhaps producing serious and lasting injury to the vesical sphincter. A perineal operation, removing the floor of the prostatic urethra and allowing the isthmus of the gland above the urethra to remain, would here, I think, be attended by better results than a suprapubic operation.

Suppose, now, a second case, similar to that just discussed, but in an older man, from sixty-five to seventy years of age. What treatment is to be pursued here? If his constitutional condition be as good as was that of the patient just mentioned, I would employ a similar treatment, and advise suprapubic prostatectomy under the same conditions, but at an earlier date. If postponed too long the

health of a patient at such an age may rapidly fail, and any operation prove too late or too great a risk to save his life.

In a third case the patient may first come to us after he has been using the catheter for months or years. He may apply because of the greater frequency with which the catheter must be used, because of the greater pain and difficulty attending its introduction, or for both reasons. In any circumstance, he probably has severe cystitis and impaired kidneys. If drainage by a permanent catheter through the urethra, combined with the internal administration of urinary antiseptics, washing out the bladder, and careful diet, effects an improvement in these particulars, I would not hesitate to resort to Freyer's operation as the surest method of saving his life and restoring his urinary function, providing the prostate were adenomatous. If no improvement in the cystitis was apparent, I would at any rate open the bladder either suprapubically or through the perineum, and direct my further treatment in accordance with what I then discovered by sight and touch. If the bladder before operation appeared to be small and contracted and the prostate hard and not very large, I should open the perineum, and at least drain the bladder, and at the same time remove so much of the offending organ as the condition of the patient and the state of the parts appeared to countenance. If the bladder appeared to be dilated and the gland adenomatous, I should prefer the suprapubic route, completing the operation by Mr. Freyer's method, if possible, but at least draining.

If drainage only had been instituted, three terminations to the case present themselves: (1) The patient dies from the progressive asthenia and nephritis, in spite of the drainage, or he recovers (2) sufficiently satisfied with a permanent urinary fistula to seek no further treatment, or, finally, (3) he still requires a prostatectomy, which now, in the absence of cystitis, can be performed with little danger.

Still another case may occur: A patient, old, feeble, perhaps delirious, has chronic retention of urine, the urethra not being permeable to instruments. Hemorrhage from the urethra, and perhaps into the bladder, complicates the case. Evidently the patient is in a critical condition. Strictures may make matters worse. At all events, the existence of an enlarged prostate is definitely determined; but the history throws little light on the primary cause of the urinary retention. What is to be done? Castration, ligation of the internal iliacs, the Bottini operation, suprapubic or perineal prostatectomy are all discarded, some as futile, some as preposterous, others again, as entailing far too great shock. The only thing left to do is to drain the bladder. I do not think simple aspiration in such a case as this will be of any lasting benefit, but rather that it will do harm by relieving the tension and encouraging vesical hemorrhage, which, in turn, only makes matters worse. To relieve the

immediate distress of the patient it may be advisable to thus draw off his urine, but it is idle to hope that the obstructions in the urethra will subside soon enough for permanent drainage to be available by that channel, and, in my opinion, the surgeon had best proceed at once to a suprapubic cystotomy. Such an operation can, if necessary, be done under local anæsthesia from cocaine, and does not involve an incision into the prostate, which would be necessary in a perineal cystotomy, and which would much increase the risk of hemorrhage. The bladder being opened, any vesical oozing could be controlled with hot solution. If the patient did not react from the serious condition in which he was at entrance, his death would at any rate have been rendered easier, and he would have been afforded the only chance to recuperate which surgery has to offer. As in the former case considered, a recovery from the cystitis, with persisting debility from the enlarged prostate, would be an indication for prostatectomy; or if such an operation appeared to be too severe, a Bottini operation, which could be done through the urinary fistula if preferred, might be undertaken, with a fair probability of relief for the few remaining years of life.

TECHNIQUE OF SPECIAL OPERATIONS. *Perineal Prostatectomy.* The patient being placed in the lithotomy position, a medium-sized steel sound—one that will pass readily through the urethra—or a Ferguson's prostatic depressor, is introduced into the bladder, and a straight median incision is made in the perineum from the base of the scrotum to a point near the margin of the anus, of course not dividing the sphincter. The membranous urethra is then exposed in the usual way, and, if preferred, may be opened. The dissection is then continued, dividing the anterior fibres of the levator ani, which are drawn to each side with hooked retractors, and the sheath of the prostate thus exposed. The rectum and its overlying tissues should be pressed backward with a blunt retractor, opening up a fairly large space in the recto-urethral triangle. By the sound or Ferguson's depressor, inverted, or with a finger passed into the bladder through an opening in the membranous urethra, the prostate can now be pressed downward and outward, stretching the process of rectovesical fascia composing its sheath. When this is incised the separation of the prostate from the surrounding structures should be begun at its posterior part, and the operator should work forward. In this dissection, which is by no means as easy as the shelling out of the gland in the suprapubic operation, I have been greatly aided by the use of Murphy's hooks, a full set of which I now regard as indispensable to the proper performance of this operation. By them each lobe of the gland is in turn drawn toward the operator and separated from the bladder. The floor of the prostatic urethra may always be removed, but the upper or anterior commissure of the gland may well be left behind, since in the class of cases in which I recommend this operation there is little likelihood of its

causing any subsequent trouble. Nor will there usually be found any pedunculated outgrowth into the floor of the bladder in patients whose hard and fibrous prostates suit them for this operation.

When as much of the gland as seems advisable has been removed, and it is evident that the vesical orifice of the urethra is as low as the lowest part of the bladder, a fair-sized rubber tube is passed into the bladder through the perineum, for drainage, being stitched to the skin, and is gently packed around with iodoform gauze. If the oozing of blood be considerable and cannot be controlled by douching with hot solutions, this packing may be quite firmly applied and remain in place for three or four days. A light gauze dressing, held in place by absorbent cotton and a T-bandage, completes the operation.

On being returned to bed, the perineal drainage is attached by another tube to a bottle hanging beside the bed. The outer dressings may be changed once or oftener daily, as required; the packing should be removed about the fourth day, and the perineal tube at the end of a week. Commencing in the second week, a full-sized catheter or sound should be passed through the urethra every third or fourth day. It will commonly be found that before the end of the second week the patient will pass more urine by the urethra than through the perineal wound. He should be kept quietly in bed until the urine ceases to pass by the perineum. To encourage the voluntary passage of urine, he may turn on his side, or even into the prone position early in the second week.

Suprapubic Prostatectomy. My own technique for this operation does not differ materially from that described by Mr. Freyer.

The patient being in the Trendelenburg position and having not more than 4 ounces of fluid in his bladder, this organ is opened by a suprapubic incision of about four inches in length. The margins of the vesical wound are then to be secured to the abdominal wall by a suture of silkworm-gut on each side. These sutures should pass through the whole thickness of the abdominal wall, and may be either tied and left at the conclusion of the operation, or caught in hæmostatic forceps and removed before dressing the wound. The former plan, which makes a ventrosuspension of the bladder, is not without its subsequent advantages.

By thus securing the bladder to the abdominal wall before the enucleation of the prostate is begun, we both steady this latter organ and render urinary extravasation between the layers of the abdomen exceedingly unlikely.

A rubber catheter is then introduced into the bladder through the urethra, and an incision of an inch or less in length, parallel to the urethra is made in the mucous membrane over the most prominent portion of the prostate as it projects into the bladder. A pair of scissors is safer than a knife for this purpose. The index finger of the surgeon is then introduced into this cut and readily finds a

natural plane of cleavage of the prostate from its sheath. The index and middle fingers of the other hand, gloved, are introduced into the rectum, and with the thumb making counterpressure as well from the perineum, the prostate can be enucleated in a surprisingly short space of time, and with unexpected ease. The vesical orifice of the urethra has in my operations generally torn loose from the bladder wall, and the prostate has come away from around it as a glove is stripped off the finger, leaving the proximal inch of the urethra, formerly embedded in prostatic tissue, lying free in the bladder and only attached where it pierces the posterior layer of the triangular ligament to become the membranous urethra. I have removed the entire prostatic urethra in some of the cases, from which there have been no untoward results; in fact, I believe that it is the rule for the prostatic urethra to be removed with the prostate. The lobe first attacked should be separated from its attachments first posteriorly, then on the outer side and below, and, finally, from the triangular ligament at its apex. If the gland be not too large, both lobes can be freed and delivered through one incision; but if it be of unusual size, it will be found advisable to repeat this procedure for the second lobe through another incision, whereupon each lobe may be delivered separately into the bladder. During these manipulations the original incision in the mucous membrane is, of course, torn larger by the finger.

By clinging close to the capsule of the gland, or even by passing between its outermost planes of tissue (as is claimed by some is always done in this operation), the prostatic plexus of veins is not injured, and there is comparatively little hemorrhage. If the veins are laid bare, it is evident that the whole capsule has been removed; but if a smooth-walled cavity is left behind, the surgeon's finger has, in all probability, split the prostatic capsule, and leaves a layer of this tissue in place. It makes no difference to the success of the operation which has occurred, except that in the former instance some of the veins might be ruptured and cause troublesome hemorrhage. Especially apt is this to be the case where the prostate is the seat of a fibrous rather than a glandular enlargement, or where periprostatitis has existed.

When the surgeon has removed the prostate he will be very much surprised, as well as pleased, to see with what rapidity the cavity from which it has been enucleated contracts. This occurs largely from the pressure of neighboring structures and from the natural elasticity of the parts, and is an event which diminishes the bleeding. Free douching of the bladder with hot saline solution, which both cleanses and checks the unavoidable oozing, together with the introduction of a suprapubic tube, completes the operation, which usually, from beginning to end, lasts not more than thirty minutes. The suprapubic wound may be sutured, except where the half-inch rubber drainage-tube emerges, this being stitched to the skin, and

is dressed with plenty of gauze and absorbent cotton, into which the tube discharges. A catheter is not retained in the urethra, nor do I drain through the perineum. It is sufficient to pass a rubber catheter by the urethra once in every twenty-four hours after the first day or two and to douche the bladder through it, allowing the fluid to drain by the suprapubic wound; recently, however, it is not my practise to wash the bladder out, except through the suprapubic wound.

After forty-eight hours the suprapubic tube may be removed and the patient encouraged to pass his urine. Voluntary micturition commonly returns earlier after this operation than after that by the perineal route; and, as there is no fear of a sinus persisting below, the patient may be propped up in bed on the fourth or fifth day, and be allowed to sit in a chair at the end of a week or ten days if his general health permits.

In both these operations the power of procreation is, of course, destroyed by the unavoidable injury to the ejaculatory ducts; but, as it is questionable whether the spermatic fluid would be ejected without the contraction of the prostate, and as it is probable that it would not be fertile without the secretion of the prostate, the destruction of these ducts is a secondary consideration. The power of intercourse is less apt to be impaired after a suprapubic enucleation than after a perineal prostatectomy, and in some patients in whom it has been lost prior to operation, this procedure appears to have restored it.

A brief report of the cases, 13 in number, in which I have had to resort to Freyer's operation, is appended.

I wish to take this opportunity of crediting my friend, Dr. Astley P. C. Ashhurst, with much of the work done upon this paper, and in particular with the bibliography.

All of these cases were under my care in the German Hospital:

CASE I.—T. F., aged sixty-three years; admitted July 30, 1903; discharged September 7, 1903; recovered. Present trouble began in May, 1902. Patient had pain upon urination, had trouble in starting the stream, then a sudden stoppage of the stream. At this time he passed two small stones, some blood, but no blood lately. Passes urine quite often, and at night some eight to ten times. Patient says that when he is up and about he has a feeling that there is a foreign body in the bladder.

July 31, 1903, a stone was detected by means of passing a sound into the bladder.

August 3, 1903. Operation: Three stones removed from the bladder, diameter of a penny and about three-eighths of an inch in thickness. Prostate enucleated.

CASE II.—L. F. (No. 1533), aged sixty-eight years; admitted June 30, 1903; discharged September 11, 1903; recovered. No venereal

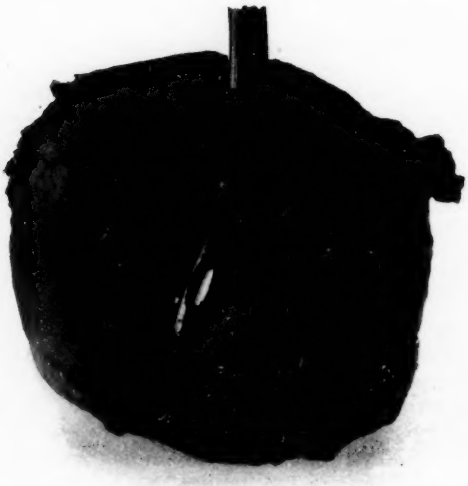
history. Bowels are regular. Moderate user of tobacco; no alcohol. One week ago infection of middle finger of left hand. For the past two years patient has had trouble in passing his urine, often obliged to get up eight and ten times at night. Some burning upon urination and difficulty in starting the flow. Four days ago inability to pass urine; catheter passed with great difficulty.

On admission 600 c.c. of bloody urine withdrawn; a false passage was detected in passing the catheter.

Rectal examination shows an enlarged prostate quite firm to the touch.

Operation, July 6, 1903: suprapubic prostatectomy.

FIG. 1.



CASE III. T. C., aged seventy-seven years. Reduced one-fifth.

CASE III.—T. C. (No. 1623), aged seventy-seven years; admitted September 19, 1903. Patient had been suffering from frequency of urination for years, more marked at night. Ten days ago urination became difficult; three days before admission impossible. He was catheterized by a physician for two days with difficulty. Third day catheterization was impossible (Fig. 1).

On admission, bladder greatly distended, reaching to the umbilicus. Prostatic catheter passed; several strictures encountered anteriorly; at the prostatic urethra is a large, false passage leading to the left.

Prostate greatly hypertrophied, the size of a small orange. Urine obtained by catheterization very bloody.

September 21, 1903. English catheter passed, permanently fixed in bladder.

Operation, September 23, 1903: suprapubic prostatectomy. Patient never rallied after operation.

CASE IV.—J. M. C. (No. 1555), aged sixty-three years; admitted July 18, 1903; discharged August 14, 1903; recovered. Patient uses alcohol moderately; tobacco to excess (Fig. 2).

Six months before admission patient first noticed difficulty in starting the stream, especially in the morning; as a rule, was com-

FIG. 2.

CASE IV. J. M. C., aged sixty-three years. Reduced one-fifth.

pelled to urinate once during the night; during the day passed urine about four or five times. Amount said to be scanty; slight pain on starting.

One week before admission was exposed to cold and rain and suffered retention; was relieved by catheter, and since then has been catheterized twice a day.

Rectal examination reveals a firm mass at the neck of the bladder, about the size of a large hen's egg. Amount of residual urine, 50 c.c.

Operation: suprapubic prostatectomy.

CASE V.—D. D. (No. 1469), aged fifty-eight years; admitted May 4, 1903; discharged June 3, 1903. Bowels are regular. Patient uses

alcohol and tobacco moderately; complains of a burning sensation after urination; about one month before admission had to urinate every ten minutes, passing 10 c.c. to 15 c.c. at a time; the stream would suddenly stop and then start up again. Urine highly colored, red, supposed to be bloody. Pain more marked on moving about.

Used to have to urinate every twenty minutes during the night; of late has not urinated so often; three or four times. Pain starts just above the symphysis pubis and shoots down the penis; stinging sensation at end of penis (Fig. 3).

Operation: suprapubic prostatectomy; recovery.

FIG. 3.

CASE V. D. D., aged fifty-eight years. Reduced one-fifth.

CASE VI.—S. L. T. (No. 1542), aged seventy-three years; admitted July 9, 1903. Patient has never used alcohol. Had an attack of gonorrhœa when about eighteen years of age Present illness began about two and a half years before admission, with frequency of urination, especially at night; obliged to get up about every fifteen or twenty minutes to urinate; often when upon his feet he would involuntarily urinate. Flow lacked force, coming in a thin stream; of late bright blood was present. At the beginning of the illness much sediment present in the urine.

Rectal examination shows an enlarged prostate, very firm, size of a lemon (Figs. 4, 5 and 6).

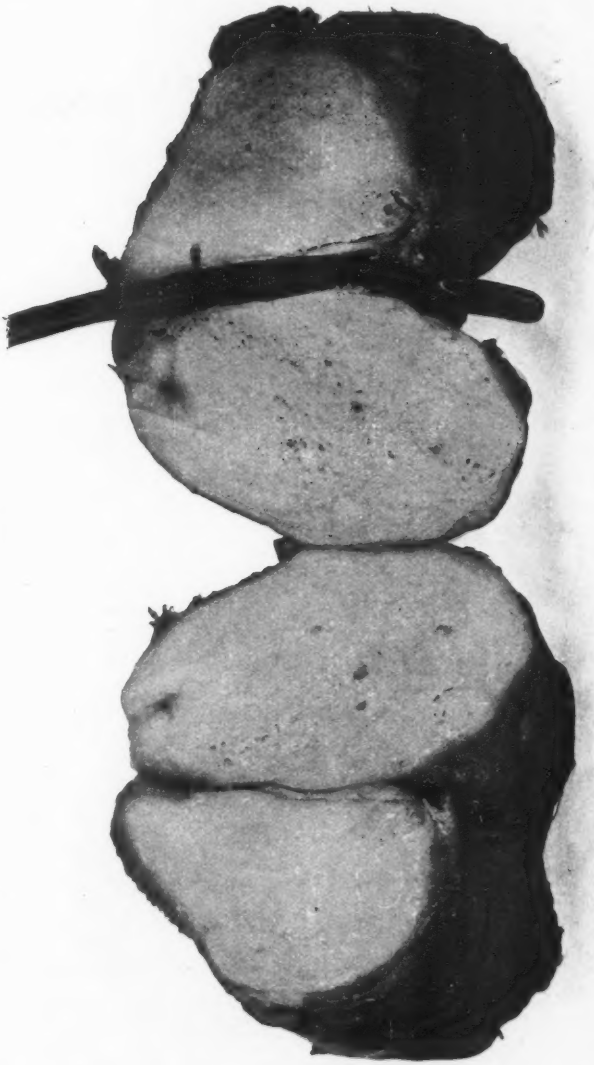
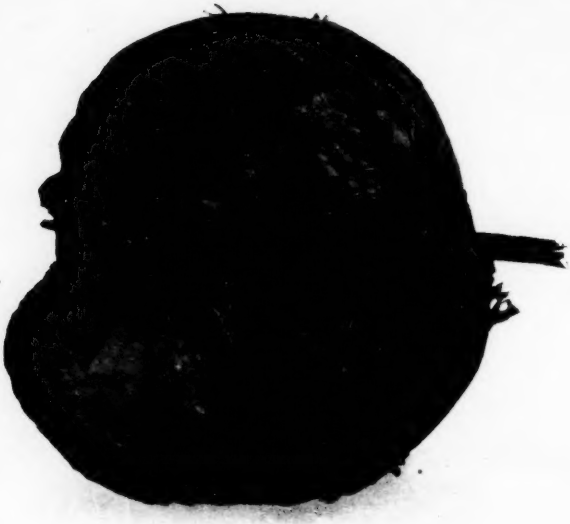


FIG. 4.

CASE VI. S. L. T., aged seventy-three years. Reduced one-fifth.

Operation: July 11, 1903: suprapubic prostatectomy. Patient died July 26, 1903.

FIG. 5.



CASE VI. S. L. T., aged seventy-three years. Upper surface. Reduced one-quarter.

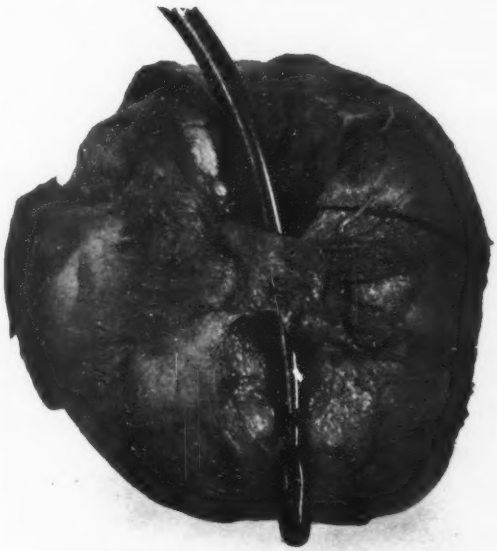
FIG. 6.

CASE VI. S. L. T., aged seventy-three years. Under surface. Reduced one-quarter.

CASE VII.—H. M. Y. (No. 1502), aged sixty-six years; admitted June 8, 1903; discharged August 1, 1903. The patient's father died of prostatic disease; a moderate user of alcohol. For past fifteen years had had frequency of urination; most marked at night. Two years before admission suffered from an acute attack of cystitis. In July, 1902, was operated upon for vesical calculus, since which time he has had a suprapubic fistula. Has not passed urine through urethra for six months.

Rectal examination shows very hard prostate, size of a lemon (Fig. 7).

FIG. 7.



CASE VII. H. M. T., aged sixty-six years. Reduced one-quarter.

Operation, June 15, 1903; a stone the size of a lima-bean extracted from the bladder; prostate enucleated; recovered.

CASE VIII.—D. R.; admitted December 9, 1901. For the last eight years patient has been troubled with dribbling of urine; for two years had had overflow of retention due to an hypertrophied prostate.

Rectal examination shows prostate size of a small orange; bilateral enlargement.

Operation: suprapubic prostatectomy. Patient died of uræmia, December 23, 1903.

CASE IX.—J. S.; admitted February 24, 1901; discharged April 5, 1901. Seven years before admission patient began to have frequent and painful urination; pain would shoot to penis and groin. Was operated two years later for stone in the bladder; both testicles became involved and were removed. Had had a perineal section done, in which part of the prostate gland was removed one year before the stone was removed. Gradual atrophy of penis. Ever since stone was crushed he had had cystitis. Urinated eight to ten times during the night and every hour in the day. Prostate hard; not enlarged.

Operation: suprapubic prostatectomy; recovered.

CASE X.—J. J. M., aged sixty-nine years; admitted September 14, 1903; discharged October 15, 1903. Six years before admission patient began to have "bladder trouble," frequent urination; for two years had used a catheter; now every two hours, with much pain.

Urine very cloudy and ammoniacal odor. By rectal examination a large soft prostate can be felt.

Operation: suprapubic prostatectomy; recovered.

CASE XI.—J. S., aged fifty-six years; admitted March 19, 1903; discharged May 9, 1903. Patient uses alcohol moderately; had chewed and smoked to excess until within ten years before admission. No venereal history. Bowels regular. Four years before admission patient noticed trouble in micturition, slowness in starting the stream, frequency of urination at night; no pain. Used catheter three to five times daily for three years. Has no difficulty in passing catheter, and when introduced expulsion from the bladder seems unimpaired.

Rectal examination reveals enlarged prostate, moderately firm, extending posteriorly into the bladder.

Operation: suprapubic prostatectomy; recovered.

CASE XII.—A. S. (No. 1826), aged sixty-eight years; admitted March 25, 1904. Patient always enjoyed good health, lived a very active life.

Present History. Had frequency of urination fourteen months prior to admission to hospital, during which time he had used a catheter as often as every fifteen minutes. Residual urine not noted.

Has been confined to bed for the last three months with continuous catheterization (Figs. 8 and 9).

March 26th. Operation: suprapubic prostatectomy. Suprapubic drainage with half-inch rubber tube. Urine from bladder through drainage-tube clear.

29th. Tube removed. Drainage from suprapubic wound without tube.

April 6th. Voided small amount of urine by urethra. Patient still under treatment.

FIG. 8.

CASE XII. A. S., aged sixty-eight years. Under surface. Reduced one-fifth.

FIG. 9.

CASE XII. A. S., aged fifty-eight years. Upper surface. Reduced one-fifth.

CASE XIII.—P. J. (No. 1810), aged sixty-five years; admitted March 5, 1904. Patient has had frequent urination day and night for some time. Four weeks before admission patient was unable to pass urine; since then he has had to be catheterized, twice a day at first, lately three times a day. By rectal examination prostate is found enlarged. Residual urine 300 c.c. (Fig. 10).

FIG. 10.

CASE XIII. P. J., aged sixty-five years.

March 12*th*. Meatotomy.

20*th*. Suprapubic prostatectomy, suprapubic drainage by rubber tube.

23*d*. Urine by tube clear.

24*th*. Tube taken out.

25*th*. Small amount of urine passed through urethra. Patient still under treatment.

STATISTICS.

Suprapubic permanent drainage.

Operator.	Cases.	Deaths.	
McGuire	39	2	
Poncet	35	0	
Horwitz	33	0	
	107	2	Mortality 1.87 %

Bottini operation.

Operator.	Cases.	Deaths.	
Bangs	42	3	
Horwitz	33	0	
Meyer	59	7	
Young	41	3	
	175	13	Mortality 7.5 %

(Freudenberg, in 1900, collected 753 operations by Bottini's method, 44 of which, or 5.8 %, terminated fatally.)

Perineal prostatectomy.

Operator.	Cases.	Deaths.	
Albarran	35	1	
Deaver	5	2	
Ferguson	6	0	
Horwitz	7	3	
Murphy	8	0	
Syms	9	0	
Verhoogen	3	0	
	73	6	Mortality 8.2 %

Freyer's operation.

Operator.	Cases.	Deaths.	
Barling	10	3	
Freyer	51	5	
McRea	3	0	
Moynihan	12	1	
Stoker	3	0	
	79	9	Mortality 11.3 %

McGill's operation.

Operator.	Cases.	Deaths.	
Armstrong	9	4	
Fuller	5	0	
Horwitz	5	1	
Thorndike	9	1	
	28	6	Mortality 21.4 %

(Belfield, in 1890, collected 88 cases of suprapubic prostatectomy, with 12 deaths, a mortality of 13.6 %; and Moullin, in 1892, collected 94 cases with 19 deaths, or 20.2 % mortality.)

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THE CLINICAL VALUE OF THE AVERAGE ANALYSIS OF GASTRIC CONTENTS.*

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of New York.

I have been led to a consideration of this subject by reading a passage in Hemmeter's[1] book on *Diseases of the Stomach*, in which he says: "It would be a valuable contribution by various gastro-enterologists of the United States if they collected and reported the frequency of hyperacidity and other gastric diseases occurring in their localities, so as to throw light on the influence of race, climate, and geographical distribution," etc., and further, by reading a statement by Riegel,[2] that the determination of the frequency of hyperchlorhydria will remain impossible, so long as every dyspeptic is not examined by lavage, and that hyperchlorhydria is encountered more frequently in private practice than in hospitals, because the symptoms of this condition are generally not severe enough to send the patient to an institution. It is evident that there is much truth in both of these apparently opposite statements, but of the two, the one which appeals to us most in connection with the subject in hand is that of Riegel, for it demands a thorough examination of each case in as scientific and uniform a manner as possible. So long as the various observers in different parts of the country do not employ the same methods and base their conclusions upon uniformly imposed conditions, so long will it be impossible to correctly determine the actual prevailing distribution and frequency of chronic gastric diseases.

We have but to glance at the reports of various observers in different parts of the world to see the force of these statements. If we take hyperchlorhydria as an example, it will be found that, so far as its frequency is concerned, it is reported as follows: by Schneider[3] (Zurich) at 5 per cent., by Kovesi[4] (Budapest) at 30 per cent., by Johnson and Böhm[5] (Stockholm) at 36 per cent., by Jaworski[6] (Lemberg) at 50 per cent., of all chronic gastric diseases; by Mathien, Bouveret, and Renaud, as exceedingly frequent in France; by various American authorities at about 50 per cent. of all stomach cases; and by Fleiner[7] (Heidelberg) and Westphalen[8] (St. Petersburg) as comparatively rare.

There can be no doubt that the different results which come to us from Continental observers may be due, in part, to differences in nationality, temperament, class conditions, and especially food, but it is also probable that they are due to non-uniform methods of

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examination, and a divergence of the imposed conditions for judging this complaint. This latter fact is especially pertinent to this country, for differences of nationality and temperament are, to a certain extent, blended here by social conditions, customs, and intermarriage; but, what is more important still, is the fact that economic conditions are such that differences in diet due to class distinctions do not operate to the same extent as they do on the Continent. We have no potato-eating Irish peasantry or rice-eating Japanese peasantry as national factors. The general scale of wages makes it possible for most artisans and laborers to nourish themselves in a proper manner. This is more than evident with ordinary skilled labor at five dollars per day. If, then, there are differences of opinion as to the relative frequency of chronic gastric diseases, we must look for the cause of these in a non-uniformity of methods employed and a diversity of opinions as to the necessary conditions to be imposed upon a patient in order that a correct diagnosis may result.

In this connection, I have chosen the acid secretion of the stomach as the subject for inspection, simply because it is the function about which we know the most and the knowledge of which, after all, so far as the diagnosis of most chronic diseases is concerned, gives us the greatest aid. It is also in the determination of hydrochloric acid that the greatest errors in judgment are made. These errors do not relate to the actual chemical reactions which take place in the test-tube or beaker after the gastric contents have been extracted, but relate to the best possible methods for developing the acid secretion of the stomach for clinical purposes. The simple finding of such and such a quantity of hydrochloric acid at a given time in a given stomach often serves only to mislead. The real object of gastric analysis is to determine the secreting capability of the stomach under the conditions in which it finds itself. This leads to a consideration of the methods which are employed for this purpose.

The whole question resolves itself into this: Can we secure a better knowledge of the gastric-acid secretion by employing carbohydrates or proteids, as exemplified in the ordinary test breakfast of Ewald or the more complicated meat-containing meal of Riegel? Or had we better use both of these meals; or, would it be wiser to discard both of these tests as placing the organ under unnatural conditions, and examine the same under the circumstances in which it finds itself, and containing the food to which the individual has been accustomed, or which has led to his discomfiture?

It would appear at first sight to be rank heresy to make a proposition such as the last, after the years of trial which the first two methods have endured; but it is also evident that there is a certain difference between gastric analysis for scientific purposes and gastric analysis for clinical diagnosis. The proposition is further justified, when we consider that very few people take kindly to tea

without milk or sugar and dry bread, and that in a certain proportion of cases a diagnosis, which remains uncertain under the usual tests, suddenly becomes clear as day if the patient is placed on his natural diet and usual conditions of life.

In order to fully consider the question, it will be necessary to review in brief three separate lines of experiment which have been pursued for years, and which have a direct bearing upon it. They may be called the diagnostic, the dietetic, and the physiological. The first two are closely allied in that they have had for their object the study of the extreme development of the acid secretion of the stomach, and the results which have been obtained in each are directly interchangeable, for that food product which is of greatest service in the development of the acid secretion for diagnostic purposes would necessarily play an important part in the treatment of gastric conditions in which the quantity of acid is found to be abnormal. A study of the question would further be incomplete should we not take into consideration the remarkable results obtained by Pawlaw in the purely physiological line, for these have revealed the marked influence of such special senses as taste, sight, and smell upon the gastric secretions in general—factors of the greatest importance in this connection.

The value for diagnostic purposes of the ordinary trial breakfast of Ewald or the proteid-containing meal of Riegel has been variously estimated by different observers. Ewald, Boas, Leube, and Jurgensen have been the chief defenders of the first, whereas Riegel, Fleiner, Fleischer, and Rosenheim have contended for the latter. The objections to the test breakfast are its simple, non-stimulating character, that it does not appeal to the special senses, and hence does not give an adequate knowledge of the secreting capability of the stomach. On the other hand, its uniform character seems to be the quality which appeals most to those who prefer it. The chief objections to the meat-containing meal have been its non-uniformity of character and the greater difficulty in manipulating it. In this connection, Rosenheim,[9] while admitting the general utility of the test breakfast, says that the meat-containing meal is better suited to scientific purposes. Fleischer[10] contends that the trial breakfast is non-stimulating, and Fleiner[11] regards the use of the two meals as largely dependent upon the habits of the patient. Riegel[12] admits the uniformity of the test breakfast, but says that it gives an insufficient idea of the secreting capability of the stomach, for a stomach may be able to produce free hydrochloric acid with a test breakfast and yet be incapable of doing the same when called upon to digest a general diet containing proteids. Leaving aside considerations of non-uniformity of character or ease of manipulation, the whole subject seems to resolve itself into a discussion of the stimulating properties of carbohydrates and proteids to the gastric secretions, for an ordinary test breakfast, after all, is largely

carbohydrates. (The ordinary wheat of the United States, according to König, contains about 10 per cent. water, 10 per cent. nitrogenous product, 2 per cent. inorganic substances, and the remainder carbohydrates.)

Another phase of the same controversy which has been going on for years relates to the proper dietetic treatment of such abnormal gastric acid conditions as hyperchlorhydria. This was practically begun by von Sohlern,[13] in 1891, after Moritz[14] (1889) and von Jaksch[15] (1890) had declared, from clinical observation, that carbohydrates were less stimulating to the gastric secretions than proteids. Briefly glancing over the various turns in the struggle, we find that von Sohlern placed all his patients upon a carbohydrate diet and was followed in the course of years by Rosenheim, Fleiner, Justesan, and Jurgensen. In 1895 Schüle,[16] after more exact experiments upon normal stomachs, declared the free hydrochloric acid was about the same in both diets, that there was a slight increase of the total acidity in cases where proteids were administered, and that the height of digestion was prolonged in the latter class of cases. In 1897 Jurgensen[17] opposed the view of Schüle by declaring that a proteid diet directly increased the quantity of acid secreted, on account of its greater combining power. In 1898 Sorensen and Mezgar,[18] experimenting upon cases of hyperchlorhydria, expressed the opinion that in this condition the free hydrochloric was about the same in both diets. In the same year Hemmeter[19] advised the use of carbohydrates in hyperchlorhydria, on account of the danger of a greater proliferation of the oxyntic cells than already existed in this condition. In 1899 Schüle[20] admitted that carbohydrates caused less secretion in hyperchlorhydria, but still maintained that the free hydrochloric acid was practically the same in both diets, and that in normal cases there was no great difference between the two.

This variety in the results obtained can only be explained by the diversity of the methods employed and the conditions imposed. In some cases normal stomachs were utilized and in others hyperchlorhydric ones. Some experimenters employed as tests meat and water, others potatoes and water, and still others bread and beef-tea. In 1899 Bachmann,[21] realizing the vagaries of former experiments, attempted to eliminate them by administering his various foodstuffs according to their nutritive values as expressed in heat units liberated by their combustion and considered only the absorbable portion of the products in his calculations. In short, he determined the quantity of food to be administered according to the isodynamic values of Rubner. These indicate that 100 grams of bread equal 190 grams of beef, or 180 grams of eggs, or 300 grams of potatoes, or 335 grams of oatmeal, or 390 grams of milk. The water contained in each was taken into consideration and proper mixtures made, so that the isodynamic value of each combination was the same.

The experiments were conducted upon previously cleansed normal stomachs, and repeated until the results were constant. The results obtained relate to the free hydrochloric acid, the total hydrochloric acid, the total acidity, the time of maximum secretion, and the duration of digestion. It was found that the first exhibition of free hydrochloric acid takes place for bread in one-half to three-quarters of an hour, for beef in one to one and one-half hours, for potatoes and milk in three-quarters to one hour. Free hydrochloric acid appears one-half an hour earlier in a bread test meal than in an isodynamic one containing beef. Free hydrochloric acid was greatest in case of potatoes and lowest in case of milk and eggs. With a standard for bread taken at 100 the quantity of free hydrochloric acid secreted for beef and potatoes would be 11 per cent. to 21 per cent. plus respectively, and for eggs and milk 14 per cent. to 16 per cent. minus respectively. Such differences existed for simple foods. In cases of more complex diets, that one containing beef showed on the average 12 per cent. more free hydrochloric acid. According to the same standard, the total hydrochloric acid was found to be 5.3 per cent. plus for potatoes, 36.2 per cent. plus for beef, 14.2 per cent. plus for eggs, and 1.3 per cent. plus for milk. In a complex meal containing beef the total hydrochloric acid was 22.5 per cent. plus. The total acidity was 35.5 per cent. higher in a beef-containing meal. The results obtained for the duration of digestion of the various foods was about the same as that given by Penzoldt. From all of which Bachmann concludes that a vegetable diet is indicated in hyperchlorhydria, suggesting that there are two objects of treatment in this condition, namely, the symptoms themselves and the process producing them (*indicatio symptomatica* and *indicatio morbi*). All treatment should be directed to the latter, advice which had already been given by Jurgensen and Hemmeter.

From such experiments, carried on in normal stomachs under exact scientific conditions, it was reasonable to suppose that the question had been more or less definitely settled. Here, however, we again encounter the difficulties which separate the results obtained by scientific experiment and the direct application of the same for clinical purposes. In 1900 Mayer,[22] operating in Boas' laboratory, while admitting the scientific soundness of Bachmann's observations, arrived at the conclusion that before the same could be of practical value, it would be necessary to demonstrate that the prolonged use of a vegetable diet could produce a diminution of the quantity of acid secreted. Experimenting with distinct vegetable and meat diets successively upon the same cases of hyperchlorhydria over extended periods of time, he found that the free hydrochloric acid and the total acidity were increased by the continuous use of vegetables. This peculiarity Mayer ascribes to the necessary increased bulk of a vegetable diet and the consequent increased mechanical irritation of the mucous membrane. On the other hand, the con-

tinuous use of proteids in this condition can only be looked upon as a palliative measure, in so far as such a diet has a greater combining power.

Nothing can demonstrate the soundness of these views better than the experience of actual practice. Aside from the utter impossibility of feeding patients for any length of time upon distinct carbohydrate or proteid foods, the results obtained thereby do not justify the means, for, notwithstanding the possibility of increasing or diminishing the demands made upon the average stomach by regulating the diet in one direction or the other, it will usually be found that in the great majority of cases the tendency to secrete too much or too little acid will persist even though all actual symptoms of the disease have disappeared. I have repeatedly examined at long intervals the gastric contents of patients who had suffered for years either from increased or diminished acidity and who had apparently recovered the entire use of the organ, judging from their general comfort and good physical condition, only to find that the same abnormalities of secretion continued to exist. This is most forcibly illustrated in a half-dozen cases of gastroenterotomy in which it was possible to make yearly complete physical and chemical examinations, and which exhibited no changes in their secretory conditions at the end of four, five, and six years, notwithstanding the fact that the causes for the original abnormal secretion of acid had been removed by operation. The importance of this tendency is being realized more and more yearly, and the growing feeling, judging from the most recent literature, is to place patients afflicted with abnormal gastric acid secretions upon plain, bland, and nutritious diets, with less consideration shown for their actual chemical composition than for their unirritating physical properties.

It may appear at first sight to have been superfluous to enter into this review of treatment in a discussion which only involves diagnosis, but as our methods for the development of the acid secretion of the stomach are mainly dietetic, it has seemed necessary to do this in order to have a comprehensive view of the whole subject, for it sometimes happens that the differential diagnosis of obscure gastric affections must depend upon differential treatment. The differentiation between gastric ulcer with hyperchlorhydria and simple gastralgia with hyperchlorhydria can often only be made in this way. If further there exists doubt as to the proper dietetic treatment of abnormal acid conditions in the stomach, there must also exist doubt as to the best method for diagnosing them.

In every case of chronic gastric disease involving abnormal acid secretion, the object of examination is to develop, if possible, the average capability of the stomach to secrete acid and to discover any vagaries of the same which may exist. In order to do this intelligently it would appear to be necessary to place the stomach under its most natural conditions. Can we do this by giving simple

tea and dry bread? A partial solution of this problem is found in the results obtained by Pawlaw[23] by physiological experiments conducted to show the influence of real and fictitious feeding. Briefly tabulated for the purpose of reference, these would be the following: 1. The centres for gastric secretion are located in the stomach wall (splanchnic section causes no cessation of secretion). 2. The vagi serve as transmitters of mental impressions, such as appetite and control secretion in an inhibitory manner (section of the nerves causes hypersecretion during the first few weeks after operation). 3. The quantity of gastric secretion stands in direct relation to the quantity of food introduced. 4. The secretion of acid is continuous during digestion, begins four to five minutes after the introduction of food, and gradually rises and falls to and from the height of digestion. 5. Each particular food has its special acid curve, the summit of which is reached most rapidly in a meat diet. 6. Simple mechanical irritation of the gastric mucosa produces no acid secretion.

The principal impression conveyed by these results, so far as diagnosis is concerned, is that any food test for the development of the gastric acid secretion which is to give satisfactory data for judging the average functional capability of the stomach must not only appeal to appetite, inducing special senses of sight, taste, and smell, but must also be a direct stimulant to the secretory centres in the stomach wall, for Pawlaw has demonstrated that the acid secreted during the first half-hour of digestion is due to the first, and that the real contact stimulation begins later. In the words of Troller, the test must be a physiological stimulant and a physical-chemical stimulant. An interesting experiment is recorded by Pawlaw in this connection. By feeding a dog 200 grams of food he secured 135 c.c. of gastric juice. By directly introducing the same quantity of food into the stomach through a fistula he secured 50 c.c. of gastric juice (sympathetic stimulation). By fictitious feeding of the food he secured 75 c.c. of gastric juice (vagus stimulation). The sum of the last two quantities would be 125 c.c., or pretty nearly the quantity obtained by feeding the animal in the natural way. Pawlaw found further that the introduction of simple substances, such as dry bread, starch, peptone, or egg albumen into the stomach, produced very little secretion. If, however, they were masticated first they caused some secretion. Troller also found that the introduction of food into the stomach through a stomach tube in the human subject produced much less acid secretion than if the same were first masticated. On the other hand, the mastication of tasteless substances caused very little acid secretion.

If we accept these results obtained by clinicians and physiologists —and there is no reason up to the present why we should not—the ordinary trial breakfast does not fulfil the requirements of a physiological test for the acid secretion. It fails in three ways: It fails

to excite the appetite, and hence the vagus stimulation in those cases in which it is taken with repugnance. It fails to be a physical-chemical stimulant because it does not act as an energetic stimulant to the secretory centres. It fails because it places the individual in an unnatural condition of life for examination. Hence it cannot be surprising that this meal should give a variety of results in clinical work, results which in a good proportion of cases are erroneous, or, stated in a more conservative manner, results which may be correct for tea and bread, but erroneous for the average normal diet of the individual.

In order to demonstrate this, I have taken statistics from some hundreds of cases of my records by comparing the results obtained from successive analyses in the same cases. Among women, three-quarters to one hour after a trial breakfast, I found 60 per cent. were hypochlorhydric, 30 per cent. normal, and the remainder hyperchlorhydric. Upon the next examination I found that 57 per cent. had remained stationary, whereas 43 per cent. had changed as follows: of the subacid cases, 22 per cent. became normal and 2 per cent. hyperacid; of the normal cases, 14 per cent. became subacid; and of the hyperchlorhydric cases, 2 per cent. became normal. Among men the changes were not so marked. Of these cases, 43 per cent. were subacid, 35 per cent. were normal, and 22 per cent. hyperchlorhydric. Upon the next examination 72 per cent. remained stationary and 28 per cent. changed as follows: of the subacid cases, 10 per cent. changed to normal; of the normal cases, 4 per cent. changed to subacidity and 6 per cent. to hyperchlorhydria; of the hyperchlorhydric cases, 12 per cent. changed to normal and sometimes to subacidity. Taken collectively, men and women, it was found that 60 per cent. remained stationary and 40 per cent. had changed upon successive examinations.

This variation cannot be due to the uncertainty usually attached to first examinations. These were carefully excluded, as I have come to the conclusion that first examinations generally give false results. This is due to misunderstanding in regard to quantity and quality of the food to be taken, duration of digestion, and such emotional disturbances as anxiety, uncertainty, and fear. Upon the second examination these objections are largely removed. Nor can the variation be attributed to nervous dyspepsia, for such cases were excluded from the list. There remains but the one conclusion that the differences in results are due to something inherent in the test itself. This is not surprising when we recall the results obtained by Pawlaw. The average patient approaches plain tea and dry bread with disgust and annoyance. It does not appeal to his appetite through the special senses, and yet nearly one-half of the gastric secretion is induced through them. In this connection, we have the interesting observation, made by Pawlaw, Bachmann, and Buch, that the addition of a little butter to the bread, thus rendering it more

palatable, will often materially increase the quantity of acid secreted, though fats are supposed to inhibit the acid secretion. That this appeal to the appetite of the individual is of paramount importance is best illustrated in the one-time manufacture of commercial pepsin, when pigs were placed within sight and smell, but out of reach, of enticing food, only to be slaughtered after a length of time sufficient to allow the peptic glands to become filled had elapsed.

It will be asked why the ordinary test breakfast, which is so slightly stimulating, should produce hyperchlorhydria in any case. The anomaly can be explained in various ways. In some of these cases the increased acidity is only momentary, and is due to emotional disturbances, for such may induce either hypochlorhydria or hyperchlorhydria in stomachs which, as a rule, are perfectly normal. In other cases the abnormality is due to defective innervation of the stomach itself, in so far as the gastric secretion does not adapt itself to demands made upon the organ. In other cases, still, the hyperchlorhydria may be due to proliferation of the oxyntic cells, so that the introduction of any food substance into the stomach would probably cause an increased acidity. In the first two classes of cases the administration of a more palatable diet will cause the hyperchlorhydria to disappear. In the last class such administration will cause a still higher acidity, in that the secretion of acid is directly proportional to demand as in normal stomachs, the only difference being that in hyperchlorhydric cases there is an increase of gastric secreting cells. It is not always possible to demonstrate this by estimating the free hydrochloric acid alone, as the total hydrochloric acid may be increased markedly without there being any evidence of this in the quantity of free hydrochloric acid present. This latter quantity may fluctuate to a certain extent in the case of more complex diets. This is due not only to the combining power of the administered food, but also to the muscular activity of the gastric walls to secure a thorough admixture of the food and the acid secreted; and, further, to the intensity of the chemical reactions which takes place in the stomach, for from recent experiment there is some evidence that the combination of hydrochloric acid and proteid within the stomach need not be permanent, but that part of the acid may be released from the acid-proteid molecule to begin anew the attack upon other proteid molecules.

It is not necessary to conclude from these observations that the ordinary test breakfast has lost its usefulness. On the contrary, as a preliminary and comparative test, it is invaluable, for any stomach which will secrete too much acid with this test may be said to have something wrong either in its innervation or its secreting structures. In order to determine the causes of abnormal secretions produced by this method, it would be advisable to compare the results obtained by it with those obtained by other methods from which some of its objectionable features have been removed; in other

words, some test which will appeal more to the patient and which will be more normally physiological in its action.

Some of the objections to the test breakfast can be removed by the addition of proteids of some kind, as typified in the meal of Riegel. Such addition would serve several purposes. It would render the test more acceptable to the patient in that its preparation would enhance its savor. The results obtained would roughly indicate the difference between carbohydrate and proteid stimulation. In cases of subacidity after a simple test meal the increased stimulating quality of the proteid may produce a normal quantity of acid, showing that the membrane is still in condition to furnish sufficient acid for digestive purposes. Finally, in some cases of hyperchlorhydria after a test breakfast, due to the exotic nature of the latter, proteids may again produce normal results. In many cases deceiving extremes would be curtailed, and we should expect more uniform results. In order to accomplish these ends in the simplest possible manner, it has been my custom to employ a meal composed of the ordinary test breakfast, with the addition of one-quarter of a pound of lean, finely chopped beef prepared by broiling and slight seasoning to render it palatable. This meal is usually taken with relish, is easily extracted from the stomach, and filters as readily as an ordinary test meal. It is examined three hours after it is taken.

Operating in this manner upon a long series of cases, 40 per cent. were found to be subacid, 46 per cent. normal, and 14 per cent. hyperchlorhydric. Upon further examinations, 35 per cent. remained subacid, 54 per cent. remained normal, and 11 per cent. hyperchlorhydric. Compared with the results obtained after trial breakfasts, the number of subacidities was reduced 23 per cent., the normal cases increased 15 per cent., and the hyperacid cases increased 9 per cent. This would indicate that the results after the proteid-containing meal are not only more constant, but also that the frequency of the various conditions is changed, the general tendency being to bring the extremes of secretion to the normal standard. This is perhaps best illustrated in a series of cases in which both meals were employed. In these cases after several examinations the free hydrochloric acid remained stationary in 67 per cent. In 33 per cent. the free hydrochloric acid after trial breakfasts was diminished in 18 per cent. and increased 15 per cent. after proteid meals. Of the subacidities after the trial breakfast, 70 per cent. remained stationary and 30 per cent. advanced to normal after the proteid meal. In the same manner 27 per cent. of the normal cases became subacid and 64 per cent. remained stationary, judging from the free hydrochloric acid alone; 38 per cent. of the hyperchlorhydric cases became normal after proteids. In 20 per cent. of the cases anachlorhydria prevailed throughout.

Whatever these results may be worth, they at least illustrate that

a palatable proteid-containing test will often prevent errors in the judgment of the secreting power of the stomach by appealing to both sources of gastric secretion and by placing the stomach under more natural conditions. It has been my experience to find that, while on the one hand the repair of the secretory functions of the stomach when once damaged is exceedingly slow and generally impossible; so, on the other hand, the impairment of the same functions is slow also, and that many stomachs which under the ordinary clinical tests do not secrete properly, will do so provided they are given an opportunity. In tempting a stomach to secrete with simple tea and bread, we are only replacing the starvation diet, to which most chronic dyspeptics have long subjected themselves by a starvation test, and it is known to everyone what a liberal change in this direction will often do for a great many of these patients.

Aside from these advantages derived from the systematic and successive administration of these two tests for judging the capability of the stomach to secrete, there is another which should not be neglected. This relates to the possibility to suspect the pathological processes which have taken place in the secretory structures by an analytical comparison of the various results obtained by the two methods. If a stomach which is persistently subacid after trial breakfasts becomes normal after proteids, we may safely conclude that the secretory structures are normal. Without the proteid test the subacid condition may just as well have been attributed to a neurosis (provided always that there were not other signs of structural changes), for there had been no sufficient demand made upon the organ by the simple tea and bread. With such results one would also be justified in suspecting an atonic condition, in that the continuous presence of food had rendered the organ less sensitive to slight stimulation. If, on the other hand, the subacid condition remains stationary after both tests, it is quite probable that organic changes have occurred, for the increased stimulation of the proteid should have produced more acid, even in cases where a neurotic element exists. In those cases in which a subacidity after trial breakfasts suddenly develops into a hyperchlorhydria after proteids, there is fair evidence that a condition of defective innervation may be present, for were the cause of the marked difference to be found in a proliferation of the gastric cells in the sense of Hemmeter, Einhorn, and Cohnheim, we should, at least, have had normal acidity, with such slight stimulation as tea and bread affords. The same condition of defective innervation may be suspected in those cases of hyperchlorhydria after a trial breakfast which become normal after proteid meals, for there is nothing in the stimulating property of a trial breakfast to call for the increased acid secretion. If, however, a hyperchlorhydria after a trial breakfast persists or increases after proteids, we have good reason to judge the case one where proliferative changes have taken place in the mucous

membrane, for the secretion of acid has been directly proportional to stimulating properties of the food. This is especially the case if the symptoms of hyperchlorhydria occurs several hours after meals, showing that the secretion of the acid has been progressive and not regular. If, on the other hand, the symptoms and the production of increased acidity occur directly after meals and gradually disappear as digestion progresses, we may again suspect defective innervation. In these cases the secretion of acid is irregular, in that the mere presence of food is sufficient to cause a very rapid secretion of acid not regulated by demand and which soon exhausts itself through its own energy. Hence, both symptoms and hyperchlorhydria disappear during the later stages of digestion. That such rapid production of fluid, acid or neutral, may take place in the stomach is best demonstrated by those cases of nervous dyspepsia in which sudden and voluminous regurgitation of fluid occurs in individuals who have taken no fluids for some time previous and whose stomachs are not atonic.

I have given these few examples to demonstrate the possibility for analytical deduction from the results obtained by the determination of the acid gastric secretion. The mere presence of so much acid in a stomach at a given time is not sufficient to establish the digestive capability of the organ, and the older practitioners are justified, to a certain extent, when they look with distrust upon gastric analysis in general. Unless the average capability of the stomach to secrete is developed by a systematic course of examinations in some such manner as given, it would be far better to depend for diagnosis and treatment upon clinical observation and a good history. It is manifestly impossible to demonstrate the secreting power of a stomach by giving one meal or one kind of meal. It would be just as reasonable to expect a dog to secrete natural gastric juice by feeding him flour paste through a tube or by going into the rice fields of Japan to hope to secure an idea of the natural gastric secretion of the peasants by giving them test meals of unsavory and unseasoned plain meat and tea. In the one case proteids are required, in the other carbohydrates. In this country, and in our larger cities especially, people are accustomed to mixed diets, and highly seasoned ones at that; so that the administration of simple food tests will often not suffice to reveal the average natural secretion of the stomach. In order to do this properly more complicated measures are necessary, the rules for which have already been given in the clinical and physiological studies of Bachmann and Pawlaw, by directing us to begin with simple tests, and then gradually to lead up to more complicated ones by adding palatable proteids, etc. By a comparison of the results obtained, we will generally be able to secure a good idea of the average capability of the stomach to digest. At least, we will know how it acts under mild and strong stimulation. Practically this can be accomplished by giving simple tea and bread

tests at first and later adding to this same test a simple proteid, such as a quarter of a pound of finely chopped lean beef palatably but simply prepared by broiling and slight seasoning. Two each of these meals should be given upon fasting stomachs upon successive days.

It will sometimes happen, however, that with all possible care as to such systematic examination, we are unable to secure an adequate idea of the secreting capability of some particular stomach in its relation to given symptoms. The reason for this would seem to lie in the very nature of the conditions imposed. Scientific and exact they may be, but they are also more or less unnatural, and in certain individuals of sensitive temperament they may defeat the very object for which they were intended. In a certain proportion of chronic gastric cases, starvation tactics have been employed to such an extent that not only does the stomach not do its work properly, but all the organs are more or less dilatory in their action. In such cases it is advisable at times to feed the patient generously, but plainly, for a few days, and then examine the gastric contents three hours after a plain but appetite-inducing mixed meal. It is remarkable to what extent this simple procedure will often aid in diagnosis by giving results which are quite different from those obtained by previous methods. It may be that the dyspeptic symptoms will be temporarily aggravated by this method, but there is a certain advantage in this also, for the nature of some forms of chronic gastric disease with remittent symptoms can often only be elicited by examination during the period of suffering. At this time any vagaries or abnormalities of secretion are exaggerated to such a degree as to make it possible to detect them easily, whereas the same may be masked in the interval. I can recall several cases of individuals in whom repeated gastric examinations revealed normal secretion, but who would occasionally be taken with very severe pain in the stomach accompanied by excessive gas formation and acid eructation. Examination at this time would show intense hyperchlorhydria with food retention. As the stomachs were perfectly normal in the intervals and the attacks would occur without indiscretions of any kind, in the midst of perfect health, it was difficult to understand the cause for a long time. Close observation, however, revealed that sudden atmospheric changes would bring on the attacks. Whether the primary condition was the hyperchlorhydria, with secondary pyloric spasm and retention or *vice versa*, it would be difficult to say. Judging from the suddenness of onset and the rapidity of development of the attacks, it would appear that a pyloric spasm was the causative factor. In none of these individuals could a general neurosis be demonstrated.

The advantage, for diagnostic purposes, of the existence of pronounced symptoms at the time of examination is further demonstrated in certain other forms of chronic gastric diseases with remittent symptoms. I refer especially to conditions of occasional periodic

atony in neurotic individuals. Cases of acute indigestion with retention due to indiscretions are not included in this category. The cases referred to are only those in which a temporary atonic condition is developed as a result of intense mental and physical depression, so commonly encountered in general neuroses. That such a localized muscular weakness in the stomach should accompany the general depression is natural, and the same disappears as the general condition improves. The duration of these temporary atonic conditions may be anywhere from twenty-four hours to days, and they are probably due to temporary loss of excitability in the ganglionic centres controlling the muscular action of the whole alimentary tract. Hence, they are not atonic conditions in the usual sense of the term, for there is no inherent disability in the muscular fibres themselves. Examined during these spells, such a stomach will contain food at a time when it should normally have been empty, and the diagnosis of atony is made, especially if a little sagging is present at the same time. Lavage is ordered, tonics are given, the diet scrupulously regulated, and the patient improves remarkably, whereas a second examination at the end of twenty-four or forty-eight hours would probably have revealed a perfectly normal stomach. Here, again, is demonstrated the necessity for repeated physical examinations in order to determine the average muscular activity of the stomach, for it is quite certain that vagaries may exist in this function also.

It has not been my object in presenting this subject to suggest improvement in the technique of gastric analysis, nor have I intended to convey the impression that the determination of the acid secretion of the stomach is the factor of paramount importance in diagnosis. This one function was selected simply because it was possible by it to judge the value of gastric analysis in general in the light of physiological research. Everyone knows that a proper knowledge of the other function is quite as important, and that all facts, whether they be elicited by chemical analysis, physical examination, or the etiology, must be carefully considered in each case. It is well known further that a correct diagnosis can be made in many instances without any gastric analysis whatever. It seems to me, however, that there is more in gastric analysis than the determination of actual diseased conditions. With these once established, there still remain to be discovered all the individual peculiarities operating in each case. That such should exist lies in the very nature of the complex processes going on along the whole alimentary tract, and is best evidenced in the wide range of the symptomatology, often of a contradictory character, observed in a given number of cases of like pathological basis.

It is in the determination of the average capability of the intact remaining portions of the secretory structures to work that the chief value of gastric analysis lies. Fleiner properly remarks that

it does not make much difference which methods we employ for this purpose, provided they are used systematically, and Trousseau long ago suggested that the correct dietetic treatment of a case of dyspepsia would often depend upon the personal experience of the individual patient.

The necessity for some generally accepted system of examination for the proper recognition and classification of chronic gastric diseases is evident from the variety of opinions which exist as to their relative frequency. For some observers the presence of a small quantity of gastric mucus is sufficient to establish a diagnosis of chronic gastric catarrh. Hence, every third case must be looked upon as such. Others regard chronic gastric ulcer as a common condition, whereas in reality all modern methods of examination have very materially reduced its frequency. Gastric dilatation will be a common disease so long as splashing below the umbilicus is deemed sufficient for its diagnosis, and gastroptosis must be very prevalent if we are to take as signs of its existence a contracted thoracic angle or the floating tenth rib of Stiller. No department of clinical medicine lends itself so readily to fads as gastrology, due partly to lack of uniformity of classification, but chiefly to the absence of recognized system in methods of examination employed and the conditions imposed for the same.

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THE GENERAL MANAGEMENT AND THERAPEUTICS OF NEPHRITIS.[1]

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I shall speak solely of acute and chronic diffuse nephritis. Time does not permit that I refer to the special management and treatment of cirrhosis of the kidney, nor, indeed, to that of lardaceous, or waxy kidney. I shall make few formal citations, and merely an occasional individual reference to others. I shall endeavor to report some of my own convictions in a very brief manner. These convictions are the result of experience and study. Personal papers from which I have again taken observed facts may be found in The American Journal of the Medical Sciences and the *New York Medical Record.*

I am conscious in what follows of imperfect and fragmentary presentation of a very important subject. I trust, however, that numerous gaps and shortcomings will be supplied by others. It must be understood that I could readily amplify many bald statements were more time allotted me, and were I unconscious of the great knowledge of the men who precede.

It is my experience, and I doubt not that of many practitioners, to be called upon only on rare occasions to treat acute nephritis, which is typical according to descriptions in text-books.

The great majority of instances of acute nephritis are grafted on a former process which has existed for some time and which has probably been caused in the first instance by harmful and irregular modes of life, or become developed during the course of some infectious disease. Many of these patients have not been aware that they were suffering from any renal trouble, and it is only when acute symptoms of more or less gravity appear and they seek medical advice that their condition is discovered. At that time and after careful inquiry and research, we shall frequently remain in reasonable doubt as to whether or not the kidneys had been diseased previously, and to what extent the fresh inflammatory process stands by itself. Fortunately, therefore, our methods of dealing with acute, subacute, and in a measure chronic diffuse nephritis are not dissimilar. Wherein differences of management and treatment exist and should be rationally attended to, I shall endeavor briefly to make evident later in this paper.

The first thing to do in all cases of nephritis in which the disease shows acute symptoms is to have the patient go to bed. It is ill-advised to allow him to lie between sheets, particularly linen ones,

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during the first few days of the acute febrile attack. Not only should he be covered on all sides with blankets, but his underclothing should be of wool. A most useful garment is a long woollen night-shirt reaching from the neck almost to the feet. Heat to the surface and of a continuous character and more than is agreeable is imperatively required. No risk even of temporary chilling should be incurred. Of course, this would be much greater in cold than in warm weather. The two cardinal principles to be kept in view are:

1. Physiological rest, so far as possible, to the diseased organ.
2. Relief of function, especially through the supplementary action of skin and bowels.

The first indication is supplied immediately in the best manner by going to bed, as I have stated, because in this way, with perfect repose of mind and body, we do not fear so much the immediate bad effects of mental occupation and muscular effort. Diet, judiciously considered and most appropriate, is the next important direction of meeting the indication. With great initial engorgement of the kidneys it is wisdom to give very small quantities of food, and solely in liquid form. Thus there is much diminished residual waste, and least irritating substances passing through a clogged-up structure. A milk diet, almost exclusively, is the one usually insisted upon during many days or weeks of the primary attack. In my judgment, this dietary should be insisted upon, but with certain limitations proceeding from common sense and experience. There are many people to whom milk is repugnant, with whom it does not agree. This is eminently true of milk in raw, undiluted form; combined with lime-water or Vichy, with the addition of a little salt, peptonized or fermented, as koumyss or matzoon, it may be acceptable to some of this number. With others of the class referred to, one must fall back upon the use of gruels and broths, jellies, and, after a few days, arrowroot, rice, or other light, amylaceous food. Among the diluents, after a day or two, water in abundance may be advantageously taken, and, when given, I am satisfied that good spring-water is especially desirable by reason of its non-irritating diuretic properties. To water may be added without injury, if the patient asks for it, weak tea, lemonade, or water from a slightly alkaline spring. If the œdema or dyspnœa is threatening, or if the urine be suppressed or in small, concentrated quantity, the bowels should be emptied rapidly by a purgative enema, or by repeated doses of salts, compound jalap powder, or calomel. So soon as the bowels have been thoroughly moved, a hot wet pack properly applied, which shall cause abundant perspiration without danger of subsequent chill or undue depression, should be insisted upon. Hot-water baths are very troublesome, are no more efficacious, and are often dangerous when given without extreme care. Hot-air baths given in bed by means of an alcohol

lamp, and hot air conducted under the supported bed-coverings through a metal tube, or according to the newer Zellerman-Sheffield system, have their warm advocates, and, in institutions where the attendants and apparatus may be readily utilized, may prove very desirable. In the care of private patients I greatly prefer the hot wet pack, for its perfect simplicity and saving of fatigue, exposure, and mental anxiety in very nervous patients. Whenever there is local pain over the loins, and especially if the urine is much diminished and of high specific gravity, while containing a considerable amount of albumin and numerous casts, dry cups, or a large, warm poultice of flaxseed, with enough mustard to redden the skin, affords relief and is useful by directing the blood to the surface and decongestioning the kidneys. After the bowels have been freely moved one or more times, high rectal irrigations by means of Kemp's tubes have in my experience promoted diuresis and neutralized toxic substances from the intestinal tract which have been injurious to the inflamed kidneys. I am of the opinion that we have, in the adoption of this later acquisition to our armamentarium, a means, when suitably employed, of very great value. I object to pilocarpine as a dangerous drug to use, as it has occasioned death several times within a few days from acute bronchial or pneumonic symptoms. In one notable case, after childbirth, in my own experience, its use induced, in a very short time, excessive secretion in the bronchial tubes, and death promptly occurred.

If threatenings of uræmia are shown by increased tension in the arteries, an accentuated aortic second sound and perhaps preliminary muscular twitchings, a few doses of nitroglycerin will often relieve the situation very rapidly. Whenever such is the case, I am inclined also to order repeated doses of sweet spirit of nitre, or to add a teaspoonful to a tumbler of water, and request the patient to drink a mouthful of it every hour or two. The nitre relaxes the arteries, acts as a diaphoretic, and is effective and innocent. In its action upon small vessels it may be regarded essentially as nitroglycerin and the nitrites. If there is a real uræmic seizure, either in the form of a clonic convulsion or of a rapidly developing comatose state, abstraction of blood, either by bleeding from the arm or wet cups over the loins, will sometimes be the only means of saving life. After several ounces of blood—*i. e.*, from 8 to 20—have been taken, it is often desirable to infuse through the veins with hot saline solution. By doing so we rid the blood, on the one hand, of a certain amount of virulent toxic material that is overwhelming it, and on the other we dilute this toxin in the most approved manner so as to allow the nervous system to support it until we can clear the organism by other efficient, though slower means. In a few instances it may be necessary to recur to inhalations of chloroform, injections of chloral hydrate by the rectum, or the hypodermic use of morphine. I have less formal objections to the use of morphine subcutaneously

in the uræmia of acute nephritis than I have in the chronic forms; still, I am ever loth to recur to it unless absolutely indicated by the extremity of the case. In certain eclamptic seizures I might advise it, especially when the seizures are frequent and I believe I have to do with puerperal nephritis, and not with acute nephritis.[1]

And yet even in these instances, and although morphine injections appear to diminish the frequency of the convulsions, when given before delivery they undoubtedly add to the risk of the child. In some of these cases I am convinced that repeated inhalations of oxygen are very valuable in avoiding the manifestations of uræmia, and in promoting its cure when fully developed. In more than one example it seemed as though just the food for the poisoned red corpuscles was being supplied. As the acute stage passes, the fever disappears, albumin and casts are diminished, and blood cells are no longer found in urine, we may become less rigid about absolute rest and diet. An egg, fresh fruit, bread, a little fish, some light puddings and green vegetables may be added to the milk dietary. Also, the patient may be permitted to sit up, or move about a little each day. For the anæmia, which is often very pronounced, some good preparation of iron is desirable in moderate doses. Of these none equals the tincture of the perchloride, either with a little glycerin, spirit of chloroform and water (Reynolds' mixture), or under the form of the well-known Basham mixture, in which it is combined with spirits of mindererus. In the same way as we require iron, oxygen, more and appropriate food, as the disease becomes subacute or chronic, so we must have good fresh air and plenty of it for these patients. Those whose means permit should seek, when otherwise possible, a warm, dry, equable climate, where their life should be passed mostly in the open air, and away from fatigue, mental and bodily. Whenever it is impossible to remove these patients from their surroundings, it is incumbent upon them to avoid cold, damp weather, and when they are unfortunately exposed to it, to be warmly clad so as to resist the effects of atmospheric changes, particularly of any draughts of air to which they are peculiarly susceptible. The albuminuria itself may persist for a long while, and, indeed, may be most difficult to overcome. We have no remedy which has any direct effect probably in promoting curative changes of diseased kidney structure, and we should expect decided amelioration, if not cure, mainly through ambient surroundings of healthful and suitable character, great and continued attention to proper dietary, the use of plenty of spring-water, suitable woollen underclothing, proper, well-prepared food, and promotion of good nutrition by the use of hæmatinics and avoidance of undue fatigue and worries. From time to time it is desirable to keep the skin active by means of the wet pack or hot-air bath, and under

[1] If the quantity of blood in the urine, in addition to the amount of albumin, is considerable, the latter diagnosis is probably correct.

no circumstances should the bowels be permitted to become at all constipated. Every effort should be made to avoid chilling of the surface, undue physical effort, absorbing mental preoccupations, imprudences of diet and régime. Secondary thoracic inflammations of acute nephritis are often very threatening and difficult to treat. They occur at a time when the patient is already depleted and would bear antiphlogistic remedies badly. Moreover, they run their course rapidly and with great severity. Moderate counter-irritation to the chest walls or more distant parts is useful, but we must beware of causing blisters. We should also be careful about remedies like turpentine, which even locally may occasion renal irritation. If despite everything the acute nephritis has remained uncured and become a condition of chronic diffuse nephritis, there are some special indications which I would further emphasize. First, in regard to the condition of the stomach and intestines. Second, in regard to the skin when it becomes tense and unduly distended despite all the means hitherto employed to promote absorption. Nausea and repeated vomiting may become, both in chronic and acute nephritis, a factor of the disease which requires our most careful treatment. After other means have been exhausted, I have occasionally found that repeated small doses of Kirschwasser have been signally successful. This water, so-called, is nothing more (as you know) than a dry alcohol containing prussic acid from peach-kernels. Chlorodyne, in repeated minute doses, diluted with a little water, has helped me at times in a remarkable manner. The diarrhœa of chronic Bright's disease is combated, as I believe, when other means fail, with occasionally surprising good effects by means of prolonged intestinal irrigation with hot saline solution.

When œdema to an excessive degree exists and affects the lower limbs, in order to prevent the breaking of the skin and resultant ulceration, it is desirable to use the lancet and make several punctures, or incisions, under antiseptic precautions. Subsequently, the limbs should be frequently washed with some mild disinfectant solution and bandaged. Occasionally it is desirable to insert a small capillary tube under the skin and leave it *in situ* for a while, after connecting it with a rubber tube of small calibre, so as to carry on continuous drainage into a vessel near the bed. Of course, such punctures, or the insertion of capillary tubes, may occasionally be followed by erysipelas or local gangrene, despite every care and precaution. Withdrawal of fluid from the pleural cavities by aspiration, when the heart is pressed upon and there is marked dyspnœa or paracentesis of the abdomen, when there is great distention of its walls, or the slow withdrawal of fluid from the peritoneal cavity by means of a small cannula, left in place several days, are at times clearly indicated and required. The use of the alkaline diuretics, such as the citrate and acetate of potash, or the

use of a subacid drink with cream of tartar and lemon-juice, is frequently very beneficial when the acute stage has subsided, and there are no longer any blood cells in the urine. The former salts eliminate themselves from the economy under the form of carbonates, and when continued for a while, unless they upset the digestion notably, may ward off the development of uræmic symptoms. Such is the judgment, at all events, of many careful observers. Whenever the heart has become dilated and weak, the quantity of urine is notably increased, the circulation strengthened, and the patient's general condition improved by means of digitalis, strophanthus and, strychnine in suitable doses. In febrile conditions, and especially among the gouty and men past the meridian of life, digitalis can rarely be given with advantage, without being protected as to its action on the peripheral arterial circulation by corrective doses of nitroglycerin. Otherwise we are apt to get a bounding, tense pulse which frequently forebodes ill for the patient, unless it be neutralized rapidly by the proper medicinal agent. A point of considerable importance, often overlooked, is to avoid, in the treatment of the pyrexia, the use of certain drugs, like chlorate of potash or salicylate of soda, whenever there is distinct evidence of nephritis, because of their well-known irritative action upon the kidneys. In the matter of diet it is wisdom in every case to watch closely the amount of albumin and number and nature of casts passed in the urine from day to day. The personal factor enters here as in the rational treatment of all disease. Thus, while in the experience of some, when the nephritis becomes subacute or chronic, albumin is stated to be notably increased by allowing one or more eggs daily to the patient, it is the experience of other competent observers that with this addition to the diet the albumin eliminated in the urine will diminish in quantity very soon, and the patient will gain in color and strength and appear to be generally improved in health. In the administration of any one of the iron salts we should ever be alive to their constipating effects, and likewise to the injurious results proceeding from overdoses, as insisted upon by Tyson. Attention should be directed to the fact that in the infectious diseases, such as eruptive fevers, diphtheria, typhoid fever, pneumonia, etc., the determination of the disease upon the kidneys marches *pari passu* with the action of its toxins upon other organs. Therefore, we should not be prone to interpret the evidences of nephritis (albumin, casts, etc.) as of too threatening nature in many instances, nor change our general method of treatment much or at all, on account of the renal symptoms. When the fever abates, or convalescence is established, the renal signs and symptoms clear up as a rule, and become satisfactory in the same way and according to the same lines that other morbid manifestations of the special infection do. Of course, there are instances in which the symptoms are specially threatening from the renal standpoint, and not infrequently

I have noticed symptoms which I have felt convinced were clearly uræmic in nature. While such cases, if unobserved, or ignorantly treated, might go from bad to worse and result fatally very soon, when the symptoms are intelligently treated they usually improve by degrees, always presuming that the kidneys were normal prior to the *début* of the infection. In this connection I would direct attention to the grave form of typhoid fever beginning with nephritis and intense gastrointestinal symptoms. Here we should not be misled as to the seriousness of the infection from every point of view, the renal condition being included, of course, with the rest. In all cases of prolonged albuminuria following acute nephritis great care against cold, exposure, and imprudences of diet, as well as against overwork and fatigue, must be exercised so as to prevent, if possible, the development of chronic nephritis. If, despite all precautionary or prophylactic measures, chronic nephritis develops, we should be especially solicitous about all subacute complications; and whenever the urine shows, by increase of albumin or casts, and especially by the presence of blood casts or free blood cells, this condition, the patient should be put to bed and treated essentially as already indicated. Chronic nephritis is frequently an incurable disease, and in any event we should not expect too much from the purely medicinal measures. Some cases do eventually recover, but when this is the case, the cure should be attributed rather to the continued close observance of hygienic rules than to the influence of remedies. The best that we should usually hope for is the improvement of the kidneys functionally as well as of the work of the organs secondarily affected. Of course, as far as may be, it is desirable to treat the cause of the chronic nephritis, but frequently this is difficult or impossible, and the disease is already well established when we first recognize it. However, whenever syphilis, malaria, or suppurative processes are present, we should not ignore the indications which proceed therefrom. We must, however, always bear in mind that many of the drugs employed to relieve those conditions are inimical to the healthful action of the kidney, and may indeed increase albuminuria and hæmaturia. Notably this is true of many of the so-called disinfectant remedies, such as carbolic acid, salicylic acid, and the salicylates, sulphocarbolate of sodium, etc. Therefore, even when we are warring against the autointoxications of gastrointestinal origin, we should bear these facts in mind, and likewise the fact that with any reasonable or non-poisonous dose of these drugs it is extremely doubtful whether we can entirely destroy infective micro-organisms which go to the kidneys from the stomach and bowels, or originate, indeed, within these organs.

While it is relatively easy to point out the dangers and drawbacks of unwise medication, it is difficult to say what medicinal remedies are useful in ameliorating or curing the disease. Chronic nephritis

comes on so gradually, so insidiously, and withal is typical of such a low grade of inflammatory action, that we know of no drugs to which we may recur with hopeful trust as to their real efficacy. It is doubtful in many instances of chronic nephritis whether it is wisdom to keep the patient closely confined either to his bed, his room, or the house. Too close confinement, even though theoretically the renal functions are thus relieved, will finish by diminishing appetite and undermining strength. Therefore, when the nutrition is fairly maintained, the strength sufficient, and the dropsy only moderate, it is better to advise gentle exercise in the open air whenever the weather is favorable. Under other circumstances, and especially whenever an acute complication has arisen in any organ, close confinement to bed, or one's room, should be insisted upon during its duration.

Fürbringer has shown the wisdom of the former counsel, and where the skin was acting freely, in one notable case. Whenever the disease itself presents atypical features, or any particular symptom is prominent or threatening, special indications of treatment may arise. It is well to remember in all such cases, that while in appearance we may be dealing with or caring for only a single manifestation of chronic nephritis, we may in reality be dealing rationally and successfully with the disease considered in its entirety. Thus, for example, in treating excessive albuminuria or dropsy, diminution in quantity of urine, sudden amaurosis or uncontrollable vomiting, we may be doing our best work for all the symptoms and conditions involved in the special case of chronic nephritis. Among remedies which have been vaunted to increase the quantity of urine in chronic nephritis, without, however, notably diminishing the amount of albumin, is iodide of potash. Ringer insists upon its efficacy in this direction independently of a syphilitic causative factor in the case. Further, he insists that in many instances the amount of iodide taken daily so as to secure its best effects must be increased to 100 or even 200 grains. I have never given such large doses, nor should I be inclined to do so except in very rare instances. The majority of patients affected with chronic nephritis have a very sensitive stomach and bowels, and the iodide, even in moderate doses, is apt to derange them. Further, its unpleasant after-taste, particularly, no matter in what manner we administer it, is apt to cause considerable annoyance and diminish appetite to a marked extent. The question of the amount of albumin eliminated daily is closely related to that of the proper diet for the patient. Some physicians hold that the dietary should be of the sort which shall not tend to increase the amount of albumin lost, as they believe the loss of strength of the patient is mainly due to this fact. Others with, as I believe, equally good reasons hold that it is far wiser to compensate for this excessive drain of albumin from the blood by increasing the quantity and variety of the food,

and thus maintaining the patient's strength and vigor. It seems to me that much must depend as to our guidance upon the individual case. If headache, malaise, interference with normal digestive processes are evidently occasioned by increased dietary, it should be abridged considerably. If, on the contrary, there are no unpleasant and untoward sequelæ other than the large daily excretion of albumin in the urine, and if, moreover, the patient's general health and strength seem satisfactory in view of the nature of the case, it seems the part of good judgment to persist in a more liberal dietary. Of course, if in the course of chronic nephritis acute hyperæmia should occur, and blood again appear in the urine, it is obviously and formally indicated to return to a liquid diet, mainly of milk, until all acute symptoms have again disappeared. It is objected to the increase of diet in chronic nephritis, that there are some instances on record in which long persistence in a milk diet had apparently cured the disease. George Johnson, for example, has cited a case in which this result followed after a period of six years.

In general, nephritic patients are better off without alcohol in any form—still there are exceptions. In men and women of middle life who have been accustomed to take a glass of wine or a little whiskey and water at meals for many a long year, it is unwise, as I believe, to intermit the habit. By so doing their appetite and digestion would be impaired, they would take less food, and, moreover, become introspective, morbid, or depressed.

In regard to the choice between spirits or a good, sound wine or light beer, I believe it may be left often to individual fancy or desire. The essential matters are that stimulants should be taken, as a rule, only at meals, in small quantity, and diluted. Of course, if there has never been the habit of taking alcohol in any form with the meals, it is unnecessary and may be harmful to begin it. In instances in which there is decidedly lowered heart power, general weak circulation, and notable exhaustion, small quantities of alcohol, both as food and stimulant, should be given for a time and the effect upon the renal condition may be considered secondary to other major and imminent considerations. Occasionally, no doubt, the amount of albumin diminishes during the period the patient is taking some astringent drug. It always seems to be a *post hoc, ergo propter hoc* proposition, and I am usually of the opinion that the dietetic and other general rules laid down and observed have been more useful than the remedy employed. It is certain, in any case, that when the astringent causes inappetence or stomachal distress it should be intermitted. Even in those cases in which an acute process has intervened and there is quite abundant hæmaturia, I am confident light diet, warmth in bed, and absolute physiological rest as far as may be to the diseased kidneys have played a far more important and beneficial role than the astringent drug. Just

as in interstitial nephritis our greater fears come frequently from dangerous uræmic manifestations, so in chronic diffuse nephritis at a later stage the presence of large dropsical effusions is often a source of much anxiety. In the lower limbs or in the peritoneal cavity they occasion much discomfort, but they do not threaten life by direct interference with heart action, as large effusions of the pleuræ sometimes do. I have already alluded to the different medical ways of relieving this condition, their indications and objections. To repeat: they are hot-air baths or wet packs, possibly the use of pilocarpine in minute doses hypodermically, hydragogue cathartics and alkaline diuretics. Whenever all these fail and the condition of the patient is imminently threatening we make use of surgical procedures—*i. e.*, puncture of lower limbs, paracentesis of abdomen, or aspiration of pleura, according to what seems first of all required. In the most severe dropsical conditions I have many times tried all these seriatim. Uræmia, especially the chronic form, not infrequently occurs in chronic nephritis in the later stages when the kidney is contracted. It should be treated very much as we do the dropsical symptoms, barring the surgical procedures just mentioned. In acute uræmia when convulsions appear, purgatives and the hot pack may be employed. Chloroform inhalations may also be used immediately to shorten the attack. I am not convinced, however, despite much testimony to the contrary, that they will accomplish this result. The use of morphine in the treatment of uræmic convulsions is still a moot question before the profession. The late Dr. A. L. Loomis cordially supported its use. Professor Wm. Osler is to-day a warm advocate of its employment, and holds that he has never seen it do harm. Delafield, Musser, and Tyson endorse its use in certain cases, but restrict its indications somewhat. Tyson believes it to be particularly harmful in interstitial nephritis.

On the other hand, George Johnson and Tirard are unqualifiedly opposed to its use in chronic nephritis in either form, and claim that as a result of hypodermic injections of morphine the convulsions become more frequent and more severe. For my part, I am of this opinion in those instances in which the convulsive seizure is accompanied with the *contracted pupil*. Whenever the pupil is *dilated*, or even of *normal size*, I have seen small or moderate morphine injections of evident service. In the other instances I have seen it act in a similar way to that pointed out by Johnson.

In this connection I would add that I am much pleased to note that Holt, in the last edition of his admirable book on *Diseases of Children*, emphasizes the indication which proceeds from the size of the pupil in the use of morphine in uræmia. Morphine injections may indeed be given and promptly followed by the arrest of a convulsive seizure. Yet too often this arrest is but the prelude of a rapidly fatal ending. Perhaps this sad result might be averted, as suggested by B. K. Rachford, if immediately after the use of

morphine blood be withdrawn from a vein and followed by intravenous saline infusion.[1]

As a rule, where there is much sleeplessness, I prefer the bromides to chloral, as in cardiac weakness the latter drug is invariably dangerous. Whenever the condition is imminent and there is high-tension pulse and a fairly vigorous patient, venesection, followed by intravenous infusion with hot, sterile, saline solution, will relieve acute uræmia at times as nothing else will. I have seen almost miraculous cures from this procedure which I have had occasion to have done for me many times. There are numerous occasions when I prefer high rectal enemata to intravenous infusions. My experience with subcutaneous injections of saline solutions is not at all favorable. They are far more painful than is stated, and their immediate or remote effects upon the uræmic poison have not appeared to me as desirable as those derived from infusions or enemata.

One very important precaution in the use of saline infusions is to guard against dilatation of the right heart, a complication to which Tyson and I have directed attention. The indications proceeding from the heart and pulse in chronic uræmia call often for a heart stimulant, and digitalis and strophanthus are frequently used.

During many years and in some of my worst cases of weak heart relaxed arteries, dropsical effusions, and insufficient urination, I have found Trousseau's diuretic wine invaluable. As is well known, it is composed of digitalis, squills, juniper berries, acetate of potash, and white wine.

We are sometimes compelled to stop all medication by the mouth in view of uncontrollable vomiting. Life is then supported almost solely, at times, with nutrient and other enemata. What I have written applies in general terms to the management and treatment of chronic nephritis in childhood as well as in adult life, except that hot tub-baths may be given more easily and safely to children than to men and women. With children the undue use of hydragogue cathartics on account of their weakening effects should be specially avoided. On the other hand, they bear repeated doses of mercury in nephritis perhaps better than adults, and run less risk of salivation. This accident, as we know, should be dreaded with the latter, on account of their extreme susceptibility to the physiological effects of the drug (Roberts).

In view of late investigations by Widal and Javal, it would seem as if it were most desirable to suppress entirely the use of salt in the dietary of patients affected with acute or chronic nephritis whenever the dropsy is considerable. These experimenters have also shown the great value of theobromin as a diuretic to diminish albuminuria, and to benefit the general condition of the patients. The inferiority

[1] Medical News, March 15, 1902.

of digitalis, nitre, and squills in these regards is insisted upon. "A mixed diet, the authors agree, without salt, may safely be given to nephritics."[1]

Finally, Dr. McVail, of Glasgow, reports two cases, one of convulsions, the other of coma, in which successful results followed spinal puncture when other means failed. In his opinion these instances prove that convulsions and coma in acute nephritis are due frequently "to the sudden increase of intracranial pressure, and not to uræmic poisoning of nerve centres."[2]

PALSY OF THE EXTRAOCULAR MUSCLES IN EXOPHTHALMIC GOITRE.[3]

WITH THE REPORT OF A CASE.

By William Campbell Posey, M.D.,
of Philadelphia.

In addition to the usual ocular signs which constitute part of the cardinal symptoms of Graves' disease—*i. e.*, the exophthalmus and the so-called Graefe, Stellwag, Dalrymple, and Möbius signs, and which are dependent upon various inco-ordinations and weaknesses of certain parts of the nervous mechanism of the eye, actual palsy of one or more of the extrinsic ocular muscles is sometimes met with. The notes of a case of this nature are appended.

Sarah L., a housekeeper, aged forty-three years, was brought to the service of the writer at the Polyclinic Hospital by Dr. O. C. Heffner, of Pottstown, Pa., in October, 1902, upon account of poor sight of several months' duration. The patient had been in rather poor health for ten years or more, suffering from palpitation of the heart and nervousness, but had had no prolonged or serious illness other than attacks of muscular rheumatism. Her appetite was good and her bowels regular; she still menstruated regularly, though she had always been subject to more or less uterine trouble. She had had seven children, five of whom were living and well; she had had no miscarriages. She said that her sight had always been good until two months before, when she suddenly saw double, the double vision persisting without remission ever since. Though dizzy at times, she did not complain of nausea, nor was she subject to attacks of vomiting. She had suffered considerably from supra-orbital and temporal head pains for several years, which were at times quite violent.

[1] New York Medical Journal, November 7, 1903; and La presse médicale, October 7, 1903.

[2] British Medical Journal, October 24, 1903, p. 1054.

[3] Read before the Ophthalmological Section of the College of Physicians of Philadelphia, January 19, 1904.

She was not aware of ever having had a swelling of the neck. Dr. Heffner had noticed a prominence of the eyes, especially of the left, for several months previously. Upon examination both eyeballs were found to be slightly proptosed, the left being slightly more so than its fellow. As shown in the accompanying photograph, the palpebral fissures were widened, there being about 2 mm. of white sclera showing between the upper lid and the superior border of the cornea in both eyes. It was noted that the patient fixed with the right eye, the left being rotated somewhat downward and out-

Exophthalmic goitre, showing exophthalmus, the Dalrymple sign, and paralysis of the external, superior, and inferior rectus and inferior oblique muscles of the left eye.

ward. The Graefe lid sign was at times present in both eyes, but at others, though carefully searched for, it could not be demonstrated. When the patient was asked to follow the examiner's finger in testing the excursions of the eyes, it was found that the left eye could not be rotated above the median line; external and internal motion also was restricted; downward movements were good. All of the motions in the right eye were performed normally. The pupils were 3 mm. in size and the irides responded equally and freely to light and accommodation stimuli. The ophthalmoscope showed about 1 D. of hypermetropia in each eye with some astigmatism. The optic nerves were somewhat gray, but there was no

neuritis. The retinal veins were rather fuller than normal and pulsated upon the disk only. Vision equalled 5/6 in the right eye and 5/9 in the left. There was no restriction of the visual fields.

From a study of the double images, it was apparent that there was a palsy of the left sixth nerve and of that portion of the third nerve of the same side which supplies the inferior oblique and the superior and inferior recti muscles. The patient was referred to the nervous clinic of the hospital for further study, where Dr. Spiller examined her, with the following result: "Patellar reflex exaggerated upon each side. Ankle clonus not obtained on either side. Achilles reflex in Babinski position distinctly exaggerated on each side and equal. Babinski reflex doubtful on each side, toes not moving distinctly in either direction on irritating sole of foot, tendency being to extension. Resistance to passive motion in lower limb not diminished in leg or thigh on either side. Sensation for touch and pain normal in all parts of body. Grasp of hand normal on each side. Triceps tendon reflex, biceps tendon reflex, and wrist reflex exaggerated and equal on each side. Gait normal. Station erect, normal, even with eyes closed. Motion of upper limbs normal in all directions. Resistance to passive movements at elbows normal. Tongue protruded straight in median line and movements free in all directions; no marked atrophy; no fibrillary tremors. Movements of facial muscles normal on each side. Patient has difficulty in drawing either corner of mouth upward separately, but in showing teeth the muscles of mouth are drawn up well. The eyelids are closed normally and the forehead wrinkled properly on each side. Masseter contracts firmly on each side. Sense of smell seems to be intact. Hearing seems to be good on both sides. Pulse 110 to 120. Slight prominence of thyroid."

An examination of the urine showed specific gravity 1026; no albumin or sugar.

A diagnosis of Graves' disease with the unusual finding of palsy of the extraocular muscles was made, and with the concurrence of Dr. Spiller the patient was advised prolonged rest in bed and specific treatment. This was adopted, but after a few weeks' trial the patient tired and left the hospital without any apparent change in her condition.

Instances of palsy of the extraocular muscles in Graves' disease are rare, but a search through the literature reveals a number of cases somewhat similar to the one just reported. Quite lengthy reference to these may be found in several interesting monographs, of which the most notable are by Ballet,[1] Liebrecht,[2] Buschan,[3] Mannheim,[4] and Möbius.[5] It would appear, from the cases reported and cited by these authors, that the paralysis may affect a single

[1] Gaz. hebd., 1888, p. 558.
[2] Klin. Monatsbl. f. Augenheilkunde, 1890, p. 492.
[3] Dissertation, Leipzig u. Vienna, 1894.
[4] Dissertation, Berlin, 1894.
[5] Zeitschr. f. Nervenheilkunde, vol. i. p. 460.

muscle or group of muscles of one eye, or even all the extrinsic muscles of one eye; and that occasionally one or more muscles of both eyes are simultaneously affected. Palsy of the ciliary muscle or iris is not recorded; nor of any of the associated movements of the eyes with the exception of reports by Schmidt-Rimpler[1] and Vossius[2] of cases of paralysis of convergence. Palsy of the levator palpebræ superioris in conjunction with palsy of the other branches of the third nerve is rare; but is rather more common without such association. The palsy may come on early in the course of the disease, but in most cases it is a late manifestation; though usually persistent, it may be but a transient condition, the muscles regaining their full strength.

The palsy may occur without paralysis of other cranial nerves, or it may be associated with a similar affection of one or more of them; thus Bristowe[3] has recorded the case of a young man who developed the symptoms of ophthalmoplegia externa three years after the ordinary symptoms of Graves' disease had manifested themselves. There was also right hemianæsthesia, with color blindness and loss of taste and smell on the same side, some palpitation and dyspnœa with headache and sickness. After a period of some months, epileptic fits came on, and later on hemorrhages from both ears. Death finally occurred from bronchitis. Autopsy was negative.

Similarly in a case reported by Warner[4] there was binocular external ophthalmoplegia with palsy of the facial and trigeminal nerves. Jendrassik[5] saw paresis of the muscles of mastication in addition to palsy of the rotary muscles of both globes, as well as most of the muscles of the face and palate. Of interest in this connection also are four cases reported by Ballet:[6] the first, the subject of severe hysteria, in addition to ophthalmoplegia externa, presented a number of bulbar symptoms—*i. e.*, palsy of both facials and paresis of the hypoglossus. In the second, also the subject of severe hysteria, there was right-sided hemianæsthesia and loss of smell and taste on the same side. Autopsy showed no visible change in the central nervous system. In a third case, in addition to palsy of both external rectus muscles, there was right-sided hemiplegia and hemianæsthesia, with epileptic attacks. The fourth case was an instance of ophthalmoplegia externa with paresis of the facial and hypoglossus. Chevalier[7] saw palsy of the externi and oblique muscles associated with disturbance in the supply of the fifth and seventh nerves and of speech.

In a case of Maude's[8] the lesion seemed to spread over the entire

[1] Nothnagel's System, 1898.
[2] Beiträge zur Augenheilkunde, 1895, vol. xviii.
[3] Brain, 1886, p. 313.
[4] Medical Times and Gazette, 1882, p. 540.
[5] Arch. f. Psych., 1886, vol. xvii. p. 301.
[6] Loc. cit.
[7] Thesis, Montpellier, 1891.
[8] St. Bartholomew's Hospital Reports, 1892, vol. xxvii,; also Brain, 1892, Autumn, p. 424.

region of the nuclei of the extraocular muscles, and those neighboring upon them, paresis of the external rectus, and probably of the superior oblique, being preceded by palsy of the facial and quickly followed by general ophthalmoplegia.

Stellwag's[1] case was one of paralysis of the lateral rotators (the ocular axes being parallel and convergence undisturbed) and later palsy of both externi with convergent strabismus and diplopia developed with transient anæsthesia and restricted movement of the left half of the upper legs.

In a case reported by Féréol,[2] of a man, aged forty-one years, six months after the appearance of the initial symptoms of exophthalmic goitre, there were headache, vomiting and dizziness, tremor, with disturbed gait and a disposition to face to the right; then diplopia, the result of right-sided trochlearis palsy. There was also right-sided diminution of motion and hyperalgesia, while on the left side there was a diminution in sensation of pain.

In a recent number of the *Deutsche medicinische Wochenschrift*, August 13, 1903, Voss, of St. Petersburg, has reported two additional cases of palsy of the extraocular muscles in exophthalmic goitre. The palsy in the first case had affected the superior and external rectus muscles of the right eye and the inferior and external rectus muscles of the left eye. There was also a partial palsy of the right facial nerve, with some cerebral symptoms.

The second case exhibited multiple lesions in the muscles of both eyes, the superior rectus muscle of the right eye being particularly affected.

The same author refers to a case of Bartholow where there was ptosis in association with gangrene and neuritis of the lower extremities.

Dyson[3] reported a case of a man with the symptoms of incipient Graves' disease, in which, in addition to slight exophthalmus and Graefe lid signs, there was slight internal convergent strabismus and dilatation of the pupil of the left eye. The patient was a chronic alcoholic.

Bruns[4] saw ptosis on the left side, with palsy of the superior recti in both eyes, with the cardinal symptoms of exophthalmic goitre, but as the patient was the subject of a chronic nephritis, it was thought that the nuclear lesion might be referable to this source and not to the Graves disease.

Wilbrand and Saenger[5] report a case with slight ptosis on the left side; with retraction of upper lid, with Graefe lid sign, upon the right side. This was explained by a bilateral affection of the nuclei

[1] Wien. med. Jahrb., 1869, p. 25.
[2] Gaz. hebd., 1889, p. 112.
[3] British Medical Journal, January 15, 1887.
[4] Anzeigen zur Zeitschrift für Psychiatrie, Bd. lx., Heft 5.
[5] Die Neurologie des Auges, vol. i. p. 47.

of the levator—*i. e.*, an irritation of the levator on the right and a palsy on the left side.

De Giovanni[1] has reported a case of unilateral exophthalmus, as a consequence of palsy of the extraocular muscles, especially of the rectus superior, in which goitre and other classical symptoms of Graves' disease were present.

In addition to these cases of more or less complete ophthalmoplegia externa, others, though more uncommon, of palsy of a single eye muscle have been reported. These have been grouped by Buschan[2] as follows:

1. Cases in which diplopia was complained of, but the muscles at fault not ascertained: Trousseau Smith, Chvostek, Stellwag, Impaccianti, Strümpell, F. Müller, and Lang-Pringle.

2. Cases of palsy of the rectus internus: West, Homén, Cohen, Westedt and Romberg, rectus interni of both eyes; Schoch and Rothmann, rectus internus and externus.

3. Cases of palsy of the rectus externus: Kurella, Mackenzie (several cases), Eulenburg, Stellwag, Makeig Jones, Liebrecht, Dyson, Ballet, and Cohen.

4. Cases of palsy of the rectus superior: Sollier, Fischer-Degranges, Kahler, Roth, Schlesinger, and Chvostek.

5. Cases of palsy of the trochlearis: Féréol.

6. Cases of palsy of the levator palpebræ of both eyes: Maude and West.

It is evident from a recital of these cases that palsy of the extraocular muscles is not of very rare occurrence and as such is not to be considered as accidental but rather as part of the morbid process of Graves' disease. Just what this part is and what the character and where the seat of the lesion may be, cannot in the still uncertain state of our knowledge regarding the nature of Graves' disease be asserted with certainty, though it would appear at least that in those cases where there was more or less complete ophthalmoplegia externa, and especially in those in which there were associated lesions in the facial, hypoglossus and glossopharyngeal nerves, that the palsy was of central origin, originating in the nerve nuclei. This reference of the palsies to an involvement of the nerve nuclei is an observation of great interest bearing upon the pathology of Graves' disease, for it is additional proof of the central origin of the affection; and while the nature of the morbid process in the nuclei which occasions the extraocular palsies is still to be determined it is probable, as the palsies are sometimes transient and the autopsies which have been made (Bristow, Ballet) have failed to reveal any visible pathological change, that the morbid process is only functional, consisting of an irritation of the centres

[1] Referat aus der Deutschen medicinal Zeitung, 1889, No. 98, p. 1143.
[2] Dissertation, Leipzig and Vienna, 1894.

by toxins which may, as evidenced by the pathological changes which occur in the thyroid gland in so many of these cases, be generated in the system as the result of some perversion in the normal action of that structure.

SKETCH OF THE EXTERNAL APPEARANCES OF UVEITIS FROM CONGENITAL SYPHILIS.[1]

By Charles A. Oliver, A.M., M.D.,
of Philadelphia.

The accompanying sketch, made by Miss Paula Himmelsbach, of this city, is so faithful a representation of the ocular changes visible externally in uveitis from congenital syphilis that it is herewith reproduced.

I first saw the patient some fourteen years ago, when she came to the clinic of Dr. William F. Norris, at Wills Eye Hospital. At that time she was a small, somewhat deformed girl of ten years of age, who, presenting all the stigmata of inherited syphilis, came to be treated for a gross attack of interstitial inflammation of the left cornea, the right one having been attacked and apparently cured in three or four months' time some three years previously.

I did not again see her until two years ago, when she reported to my own clinic at the hospital to obtain a re-correction for lenses which she had obtained in the eye dispensary of the Jefferson Medical College some six years previously, and which had been uncomfortable for about three or four months' time.

In the woman I recognized the child of fourteen years before: the same scaphoid curve of the face; the sunken nasal bridge; the cicatrices at the angles of the badly-shaped mouth; the misshapen jaw; the large protruding eyeballs, and the protuberant forehead —all made an indelible physiognomical picture. Small, active, quick and energetic, she seemed not to have developed or grown from what I had once known her; this in spite of marriage and delivery by Cæsarean section some several months previously of a living full-term and apparently healthy child.

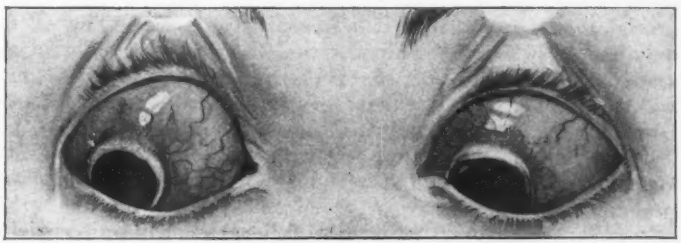
Her teeth, which were small and badly developed, gave none of the gross classical signs of configuration, though the family history, which I had obtained when I first saw her, was undoubtedly that of parental syphilis with its evil effects upon this and two other later children.

Corrected vision and accommodation were normal. Color percep-

[1] Exhibited at the March, 1904, meeting of the Section on Ophthalmology of the College of Physicians of Philadelphia.

tion was good, though possibly there were some slight uncertainty in the finer differentiations of greens and reds. The visual fields were somewhat contracted concentrically. The pupils were irregular (two by three millimetres in size); the irides, which were markedly depressed, were prompt in their various reactions. The anterior chambers were deepest in the positions corresponding with their angles and the pupillary areas. There were not any synechia nor marks of previous ones. The iris stroma was thinned, and the crypts were stretched and elongated in many places. The movements of the eyelids were normal. The action of the exterior ocular muscles was good in all of the ordinary physiological directions.

The corneæ presented some fine canalization which could be discerned only by most careful focusing. The corneal borders



External appearances of uveitis from congenital syphilis.

were densely hazed, particularly above, the dense ring-like opacity passing into comparatively clear though excessively thinned and protuberant peripheral borders. The intraocular media were transparent. The retinæ and underlying chorioids were in comparatively good condition, with the exception of a few fine inflammatory sequelæ and degeneration scars, which were situated well anteriorly and to the lower inner quadrants of the membranes. In places the ciliary regions were thinned and opalescent, but there were not any distinct ectasiæ. The anterior scleral veins, and, in places, the corresponding overlying subconjunctival and conjunctival ones, were engorged and tortuous. The eyeballs were rather large, though apparently symmetrical in their relative diameters.

ACTINOMYCOSIS OF THE TONSILS,

WITH SOME REMARKS ON THE PORTALS OF INFECTION.

By Jonathan Wright, M.D.,
of new york city.

(From the Throat Department of the Pathological Laboratory of the Manhattan Eye and Ear Hospital.)

The only report I can find in literature of the lesion in this situation is by Ruge, in 1896,[1] a member of Gerhardt's clinic. He described four cases, which, wonderful to say, he discovered by the microscopic examination of only twenty-five tonsils. He suggested that systematic examinations of excised tonsils would more frequently reveal the condition. On a reference to my index I find that I have examined microscopically hypertrophied faucial tonsils from more than seventy-five cases. This is the only one in which an actinomycotic clump was present in the sections. It is possible of course that if the whole of every tonsil was reduced to sections, something of Ruge's prediction might have been observed. The clumps, or, as the Germans call them, the "drusen," are not to be mistaken by anyone accustomed to histological work. In a search through the *Centralblatt für Laryngologie* since its first number, a matter of twenty years, only one other reference was found to the existence of a local lesion of actinomycosis in the tonsil, though abscesses and infiltrations around the jaws and neck are cited in great abundance. With the report of this case I am familiar only in references,[2] it having been published originally by Lesin in the Russian *Wratsch*. This latter case died of cerebral involvement and a focal abscess was found in one tonsil and a scar in the other. Ruge concluded, from the fact that his cases presented no lesion elsewhere and had no subsequent trouble after excision of the tonsils, they could only be something that resembled actinomycosis, and reported them under the title "Ueber Actinomyces-ähnliche Gebilde," etc.

Therefore, if rarity of occurrence is any excuse for the publication of this case, I might be justified on that ground alone, but a "rare case" which fits into no category and teaches no lesson furnishes idle reading and is a futile labor for the author. The fewer such facts a man has to put in his mental storehouse the better.

It is my interest in the suggestiveness of this case which induces me to bring it before you for consideration. Incidentally it illustrates one point which I have attempted to emphasize on many occasions, and that is the desirability of examining microscopically

[1] Zeitsch. f. klin. Med., 1896, No. 30, p. 529.
[2] Centralbl. f. Laryngologie, 1895, No. 11, p. 901; Baumgarten's Jahresbericht, No. 10, p. 454.

absolutely all tissue removed from the nose and throat in the routine of either hospital or private practice.

This case was a boy, aged twelve years, who came into the clinic of the Manhattan Eye and Ear Hospital, in the service of Dr. Lewis A. Coffin, for enlarged tonsils. They were cut out by Dr. W. N. Hubbard, one of the assistant surgeons, and the boy went on his way rejoicing, was well a month later, and still remains so, no metastasis having as yet shown itself.[1]

FIG. 1.

Camera lucida drawing of a section of a tonsil, showing under low power an abscess cavity containing masses of actinomyces. At *a* is seen the beginnimg of the growth of a colony in one of the lymph nodes. There is a larger number of lymph nodes than usual in tonsillar hypertrophy.

In the routine of the clinic these tonsils were put in formalin and later in alcohol, embedded in celloidin, cut in sections, and stained in hæmatoxylin-eosin. Subsequently the whole tonsil was sectioned, but only the one cavity was found. With the various microscopic appearances of that we are here concerned.

In Fig. 1 it will be seen the section traverses the whole tonsil covered with the usual striated squamous epithelium, with little or no keratosis. What is apparently an abscess cavity first attracts attention. It occupies a comparatively large area in the tonsil. In its cavity are seen the peculiar striated kernels which have at their

[1] I am unable to conjecture whether Ruge's cases were actinomycosis or not. In view of the variations in the toxicity of the different members of the group with the same morphology and, indeed, from the clinical histories of such cases, excision of a focus, even when only moderately complete, frequently prevents recurrence.

centre a blurred appearance and a yellowish tinge at the surface, taking the hæmatoxylin stain very faintly. A closer examination reveals the striations more marked at the edges. (The artist has drawn them somewhat schematically in this figure.) They are seen not only free in the cavity, but less distinctly along its walls, the granular masses making the picture indistinct.

Under a higher power there is seen in Fig. 2 the edge of one of these striated clumps and the peculiar branching, club-shaped rays of the actinomyces group appear, and through the blur and granulation of the interior of the clump one can make out the lace network, which is so characteristic. Around the ends of the ray or terminal filaments, which are variously described as loops and club-shaped

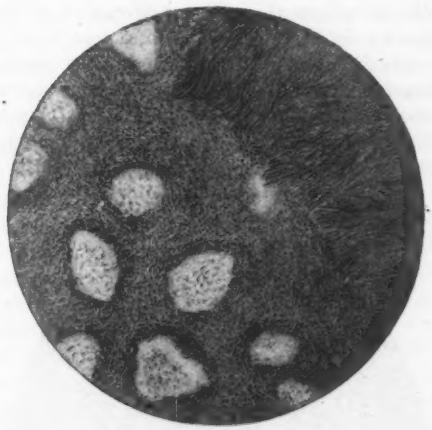
FIG. 2.

Camera lucida drawing under high power of the edge of one of the masses of actinomyces lying in the abscess cavity. The drawing does not give very accurately the "granulation" of the mass.

ends, are to be seen groups of much swollen leukocytes whose nuclei are granular or fragmented. When we come to examine the edge of the cavity we find that it is lined with a very greatly proliferated epithelium. In Fig. 3 it is seen that this proliferation differs markedly from that seen in crypts filled with the leptothrix. There is here no appearance of keratosis, but the fringe of epithelium is faint and very deep, enclosing islands of connective tissue. The nuclei of the epithelial cells are also fragmented. It has the appearance of the epithelium around an old syphilitic or lupoid ulcer. The ends of the actinomyces are pointed directly into the epithelium and invading it, apparently, as it breaks down by granular degeneration. Yet evidently the epithelium continues to proliferate under some stimulus, encroaching upon and surrounding islands of connective tissue,

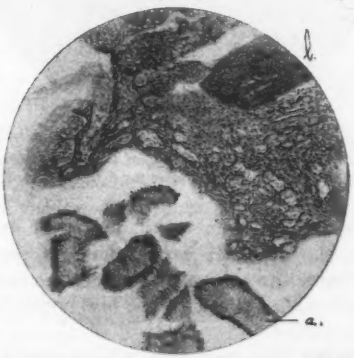
which doubtless at first contained round lymph cells. These apparently gave way first to the "granulation" process, to which the reticulum is also succumbing.

FIG. 3.



Camera lucida drawing under high power, showing the hyperplasia of the epithelium of crypt and the "granulation" due to the influence of the adjacent clump of actinomyces.

FIG. 4.



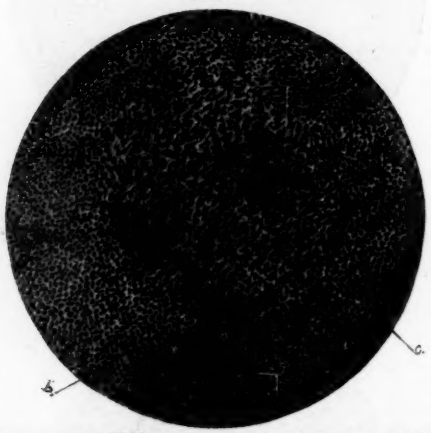
Proliferation of the epithelium of the crypt. The degeneration and the granular condition of the cells renders the epithelium atypical and indistinct. *a.* Actinomyces clumps. *e.* Epithelium. *l.* Lymphoid tissue.

It is apparent therefore that we have here the infecting germ lodged in a tonsillar crypt. After a longer or shorter time it began to multiply and excrete a toxin which, while it stimulates the proliferation of the bordering epithelium, gradually leads to its disintegra-

tion at the surface. The cavity of the crypt is thus painlessly enlarged, as there is no pressure, and the epithelium encroaches on the subjacent tissue. It is regularly the history of these actinomycotic abscesses wherever they form that they produce no pain. Neither do they form much pus, and the infection is not a mixed one. It is safe to believe that this proliferation of the epithelium has some influence in keeping the actinomyces from invading the subjacent lymphoid tissue, and we may thus understand why, without an abrasion, skin infection is such a rare occurrence.

In the warmth and moisture of a tonsillar crypt in this case this was an insufficient protection, for if you will refer again to Fig. 1 you will perceive that the lymph nodes are more swollen and

FIG. 5.



Camera lucida drawing under high power of the colony seen at *a* in the low-power drawing of Fig. 1. The fragmentation and breaking down of the lymph cells around the bundle of mycelial threads is very evident. *b*. Branching of the filaments. *c*. Edge of the lymph node.

prominent in the section than usual, and that in one of them at a distance from the affected crypt there is seen at *a* an area small in size. Under the microscope it has the same blurring and the same faint yellowish tinge with the low power as do the clumps in the crypt.

When we examine this spot with a high power (Fig. 5) we see some parallel threads, without joints, but with a tendency to branch. You will note that at *b* there is no fragmentation at the ends of the small branch, but at the terminal points of the larger and chief bundle you will note a destructive fragmentation of the nuclear material of the lymph cells. Elsewhere in the node these are normal.

A great deal has been written both as to the disease actino-

mycosis in man and in cattle, and as to the morphology and classification of the organism with which the disease is associated.[1] While it is said by Israel that von Langenbeck observed the disease in 1845, and various others from time to time referred to the condition, its nature was not recognized until the rise of modern bacteriology. Since then various names have been suggested for the organism. It has been called a streptothrix, cladothrix, oöspora, but the name actinomyces, suggested by the botanist Harz, has remained on the whole the one usually employed. MacCallum and others have used the affix asteroides to designate the appearance of the clumps, or asterisk-like "drusen." It was Bollinger[2] who first observed, in 1876, a microphyte in the lumps which are so often seen about the jaws of cattle, and referred the organism to Harz for a name. In the same year Israel described the disease in man,[3] but did not connect it with the cattle affection. This was pointed out by Ponfick[4] in 1879. Classified at first among the fungi, it was supposed to be insusceptible of artificial culture. Although Johne failed in 1882,[5] a more successful attempt was made by Boström in 1885.[6]

Since then numerous and varied experiences with its life forms and different culture media have led to much discussion.[7]

I do not propose—indeed I am not able—to refer to all the variations in the pleomorphism of the organism. Suffice it to say that it appears under various conditions as a streptothrix or streptobacillus, long threads sometimes straight, sometimes in loops, sometimes in such short rods that they resemble cocci. Club-shaped branching appearances are shown in my drawings. As I understand it, the question of spore formation has not been fully settled. Mertens[8] declares that their presence has not been proven, while the contrary is asserted by Berard and Nicolas (*ibid.*).

It seems the drift of recent opinion that "a number of different micro-organisms may cause the typical disease picture."[9] Not only is this the opinion of Dr. W. T. Howard, but more recently[10] we are to infer from the researches of the same observer, supported by the testimony of others, that there are varieties, indistinguishable

[1] A reference to the indices of the Centralblatt f. Bakteriologie, the Centralblatt f. Laryngologie, and Baumgarten's Jahresbericht will put anyone on the track of practically all that has been written on the subject in twenty-five years.

[2] Centralblatt f. d. med. Wissensch., 1877, No. 27.

[3] Virchow's Archiv, Bd. lxxiv.

[4] Berl. klin. Woch., 1879, p. 347.

[5] Deutsche Zeitsch. f. Thiermed., 1882, Bd. vii.

[6] Berl. klin. Woch., No. 1.

[7] The latest contribution I have seen being the article on the subject by Lignieres and Spitz, under the title "Contribution à l'étude à la classification et à la nomenclature des affections connues sous le nom d'actinomycose." Centralbl. f. Bakt., I. Abt. Originale, 1904, No. 3, Bd. xxxv.

[8] Centralbl. f. Bakt., 1901, No. 16, Bd. xxix.

[9] Howard. Actinomycosis of the Central Nervous System, Journal of Medical Research May, 1903.

[10] Ibid., January, 1904.

morphologically from pathogenic forms, which are entirely harmless and non-pathogenic in animals, Howard having found such fungi growing in various samples of vaccine.[1]

As yet it remains undecided whether these varieties can be distinguished by culture methods, and at present, so far as I know, observation of inoculation of animals and a study by culture methods of cases occurring in man furnish the most promising fields of differentiation. It is only fair to say that so recent a writer as Mertens[2] agrees with the earlier statements of Pfeiffer that the division of actinomyces even into two varieties seems unwarranted, but it would seem from the work of Silberschmidt (quoted by Howard) and Lignières and Spitz that this opinion is now untenable, Howard quoting Lachner-Sandoval to the effect that there are as many as twenty-five varieties of ray fungi.

As to the tinctorial reactions: "Mycelium and spores stain by Gram and retain carbol-fuchsin after treating with 20 per cent. sulphuric acid." "In sections stained with hæmatoxylin or methylene blue the colonies are finely granular, and stain homogeneously with the blue stain" (Howard).[3] On the contrary, MacCallum[4] says: "The staining is not regular or homogeneous, and one frequently finds clear unstained areas, almost like spores in some bacilli, or, on the other hand and more commonly, only granular bodies along the course of the filaments take the stain, so that the organism looks like a dotted line, sometimes with a faint outline on each side of the row of dots. These fine grains are irregular in form and do not suggest spores."

Other observers also differ in so far as they look upon some of the granules as spores or cocci forms or fragments of degeneration.

A very much more interesting and directly suggestive part of the subject is the conclusion toward which a study of its morphology and its culture has led many observers as to its classification and its affinities to other organisms. Some of its pathogenic characteristics, as we shall see later, are also, to say the least, very suggestive in this respect.

To quote again from MacCallum: "The organisms show affinities on the one hand with the bacteria and on the other with the fungi. . . . Their size approaches more nearly that of the bacteria than that of most fungi, and they stand nearer to the bacteria than to the fungi from the fact that their filaments do not, as in the fungi, consist of tubules with doubly contoured wall and divided into segments by septa. They resemble the fungi, how-

[1] It is quite possible that Ruge's cases were really the actinomyces which had failed to penetrate the tissues, owing to their feeble toxicity toward their hosts. In my own case, though growing in a lymph node at one place, the organism may have been but feebly pathogenic.

[2] Loc. cit.

[3] Loc. cit.

[4] Centralbl. f. Bakt. Originale, 1902, No. 12, Bd. xxxi.

ever, in being branched structures which produce under certain conditions fine conidia on their terminal filaments."

We may omit here any extended reference to its affinities morphologically with the Klebs-Loeffler diphtheria bacillus. The subject has received some attention from Silberschmidt[1] and others.

A much more interesting subject for consideration is its relationship to the tubercle bacillus. As long ago as 1888 Metschnikoff drew attention to the variations in the morphology of the tubercle bacillus, and Czaplewski still further elaborated the subject in 1891. Metschnikoff declared that the tubercle bacillus was not the final stage, but represented a condition in the developing cycle of a thread-like bacterium. He showed the branching of the bacillus and the bud-like projections sometimes seen even in human tuberculous sputum. Mafucci demonstrated the same appearances more often seen in fowl tuberculosis. Fischl, in 1893, showed that these forms occasionally occurred in pure cultures, and seems to have been the first to call attention to the resemblance of the bacillus in this form to the actinomyces. A. Coppen Jones,[2] from whose work I have quoted the above, in 1895 pointed out characteristics common to both the organisms, and the details are beautifully shown in his plates. Pettersson[3] in 1899 drew attention to the culture affinities of the acid-resisting bacilli, and more recently Abbott and Gildersleeve[4] have again returned to the subject and remarked upon the "actinomyces-like development of some of the acid-resisting bacilli." It must be remembered that, besides various modifications in the pathogeny of the tubercle bacillus itself, the human, the bovine, the fowl, the fish, the turtle tubercle bacillus, there are the morphologically and in cultures closely allied grass, hay, and butter bacillus, which have no pathogenic properties at all, though the above authors have shown a slight amount of tissue change occurring when they are injected. Indeed, so far as pathogenesis to man is concerned, we have in the group a steadily progressive series of organisms from the harmless butter bacillus of Rabinowitsch, through the turtle and fish bacilli,[5] to the bovine and human bacilli, concerning the mutual virulence of which war has lately been waging

We know with much less accuracy the interrelationship, as to pathogenesis, of the various members of the actinomyces group of bacilli, but we have some reason to believe that a similar series will be revealed in this family of organisms. Abbott and Gildersleeve have shown that the grass and hay bacilli injected in animals form in clumps with granular leukocytes around them in a manner similar though not identical with the actinomyces clumps.

[1] Deutsche med. Woch., 1901, No. 47.
[2] Centralbl. f. Bakt., Nos. 1 and 2, Bd. xvii.
[3] Berlin. klin. Woch., 1899, No. 26.
[4] Centralbl. f. Bakt. Originale, 1902, 1st Abt., Bd. xxxi.
[5] Friedmann, Centralbl. f. Bakt. Originale, 1903, Nos. 7 and 8, Bd. xxxiv.

Finally, the actinomyces is essentially an aerobic organism, and it has been shown by Coppen Jones, most recently supported by Wollbach and Ernst,[1] that the tubercle bacillus growing on a favorable medium shows more frequently the branched forms when the access of oxygen to the culture is most free. Whether the ray-fungus, on the contrary, shows more forms analogous to the usual forms of the tubercle bacillus when its culture medium is poor and the access of oxygen limited, I am unable to ascertain. Suffice it to say, as to classification, that generically these two microphytes seem closely related and stand between the lower and higher forms of microbic life. To use expressions current in German literature, the actinomyces is a "facultativ-parasite," the tubercle bacillus a "facultativ-saprophyte," *i. e.*, they both grow, though with difficulty, the former within and the latter without the animal body. Conversely the actinomyces grows readily on plants and the tubercle bacillus readily in animals.

It is necessary to say a word of the occurrence of actinomyces in nature and of the source whence man derives the organism when infected. It was formerly surmised, after Ponfick had shown the disease in man was identical with the more common infection in cattle, that man derived it direct from the latter, and, as usual, it was claimed in Germany that American cattle were more commonly affected than the beasts of the Rhineland. As a matter of fact, however, the majority of cases in which it was possible to trace the infection from an external source have shown that the organism was carried into the tissues, usually of the mouth, but rarely of the skin,[2] by the intrusion of foreign bodies, such as stalks and ears of grain or pieces of wood. Indeed, grain kernels have been found between the teeth of cattle, on which actinomyces were growing. It has been shown by Berestnew that straw dampened and placed in a thermostat will frequently, after a week or two and the removal of other more quickly growing colonies, show the presence of the actinomyces. The organism grows in the comparatively large air cavities and channels of the straw stems, doubtless because it is left there unmolested, yet exposed to the air a sufficiently long time to fructify and draw sustenance from the plant cells. It would seem therefore a matter of considerable doubt if the germ enters the system of man very frequently with the meat of cattle, though the number of cases of primary intestinal or peritoneal lesions might argue in favor of this method of infection. At the most it is a rare affection in man in any situation.

In discussing the port of entry of the organism we find another field in which something may be gleaned of interest in the wider topic of human tuberculosis. It is perfectly apparent, in looking

[1] Journal of Medical Research, December, 1903.
[2] Von Bergmann, Berl. klin. Woch., 1904, No. 1.

through the abstracts of cases reported in laryngological literature, that reports in the vast majority of the cases refer to lesions about the jaws and necks, many of them proved to be connected directly with a carious tooth cavity. Naturally the criticism is suggested that laryngological literature is hardly the place to seek this sort of evidence of the comparative frequency of the location of the lesion. We will therefore turn to bacteriological literature.

In 1887 Bollinger[1] stated that Moosbrugger, in collecting reports of ninety cases, found that in 10 per cent. no port of invasion could be made out, but in the majority of cases the mouth and pharynx seemed to be the starting point.

On glancing at the cases referred to in the *Centralblatt für Bakteriologie* since 1887 a tally, which may not be absolutely correct, shows that the primary lesion was situated around, or the port of entry of the infection could be traced to:

The neck or jaws, mouth or pharynx	72 cases.
Primary lesion in abdomen or its viscera	18 "
" " thorax walls, lungs, or pleuræ	21 "
" " brain	3 "
" " skin	3 "
Total	117 "

In 1899 Rütmeyer,[2] from a review of the literature surmised that the port of entry was the mouth and pharynx in 50 per cent. of the cases; lungs, 20 per cent.; intestines, 15 per cent. Of the 113 cases studied by Miller[3] in the same year, 53 were referred to the regions of the mouth and pharynx.

In a very large number of cases the tissue changes can be traced directly to a carious tooth cavity. In others they stand in such relation to a carious tooth that the port of entry can hardly be doubted. It is true many infections occur about the jaws of people with healthy teeth, but in those of untidy habits the crevices between the teeth, as we have seen in cattle, would furnish a sheltered nook for the slowly noxious effect on the epithelium. A careful analysis of the cases will show that the larger number of cases of lesions about the jaws present a lesion about the *lower* jaw, the lesions about the upper jaw being in a marked minority. We can scarcely explain this, when we have such strong evidence that the teeth are at fault, except on the assumption that the force of gravity drains the tooth cavities and normal crevices between the teeth of the upper jaw. While of course an imperfect protection, it would be sufficient to account for the greater preponderance of apparent infection through the teeth of the lower jaw. I desire to lay emphasis on this influence of gravity on the portals of infection, as I shall return to the subject when we come to consider the bearing of the

[1] Centralblatt f. Bakteriologie, 1888, p. 501.
[2] Berl. klin. Woch., 1889, Nos. 3 and 4.
[3] Die Mikro-organismen der Mundhöhle, Ed. 1889.

evidence of this rarer form of bacterial infection upon the more important subject of tuberculous infection.

Evidence of the influence of gravity is still further presented in the rarity of infection with actinomyces through the skin. This has occurred, but so far as I know the proof of its actual occurrence has, with one exception, always been associated with the entrance of a foreign body. In 1895 Hummel,[1] in a search of all literature, found but twelve cases, to which he added one, in which it seemed that the organism entered the tissues in a breach made by a foreign body, the grain of a cereal, in all but two cases. The case related by von Bergmann, in which actinomyces infection followed the kick of a horse, may be placed in the same category, but here the solution of continuity was in the skin, while in the other cases referred to the mucous membranes were pierced in the majority of the reports. While therefore we have good reason to believe that man, like cattle, receives his actinomyces infection in his food on grain and vegetables rather than in his animal food, it seems probable that the organism must either be carried at once beneath the epithelium by the intrusion of a foreign body, or it must find some lurking-place where it may remain in contact with the epithelium for a long space of time under the influence of warmth and dampness.

The query arises, Could it under such circumstances pass through the intact squamous epithelium? If you will glance at Figs. 3 and 4, you will see what effect it is exerting on such epithelium, while the evidence of Fig. 5 will convince you that somewhere in this region of the tonsil it has passed the epithelial barriers. In support of this evidence of a microbe allied to the tubercle bacillus being able to pierce squamous epithelium even of the skin, when placed under proper conditions of warmth and dampness, I may cite the instances referred to by von Bergmann where actinomyces caused lesions in the crevices of old scars, as in those from breast operations, too long after operation, presumably aseptic, to think of entrance at the time of the skin cutting. This might be a profitable point to clear up by animal experimentation, making artificial skin pockets and placing in them actinomyces of known virulence.

It is difficult to say how the chemistry of the gastrointestinal tract affects the actinomyces. It is of course certain, considering its prevalence on plants, that it is frequently carried into the stomach of man with his food, and still more frequently into the gastro-intestinal tract of cattle.[2] I am not acquainted with any investigations which show the relative frequency of actinomyces disease of

[1] Centralbl. f. Bakt., 1895, No. 2, p. 180.

[2] In this, however, I may be mistaken, for Mari (Centralbl. f. Bakt., 1892, Bd. ii. p. 854) examined for actinomycosis 42,230 cattle and found the lesion in 1030, or 0.02⅖ per cent. of the total number. Of these 1030 cases the lesion was about the lips in 621, or about 60 per cent., which as we have seen is not far from the ratio about the mouth in man. Berg (Centralbl. f. Bakt., 1899, 2te Abt., Bd. xxvi. p. 231) is said to have examined 400,000 (?) sheep and found the lesion only in three cases, and these about the mouth.

the abdominal organs of cattle. I suspect, from general laws, that such lesions are relatively rarer in cattle than in man. As we have seen, abdominal actinomycosis stands next in frequency to that of the lungs in man. It is very probable that many cases of apparent primary pulmonary actinomycosis are secondary to lesions higher up which have not been recognized, but whatever may be the truth in regard to tuberculosis, I think we can hardly thus explain the cases of actinomycosis of the abdominal cavity. In reference to this point Grill,[1] who in 1895 collected 107 reports of cases of intestinal actinomycosis, considered the germs too big to be carried by lymph channels, a statement which will hardly stand criticism to-day, though it would seem that *something* hinders them from travelling much by the lymph channels. It seems likely they are carried chiefly by bloodvessels.[2] Although no part of the intestinal tract seems to be proof against their absorption, most of the cases proceed from the cæcum and the vermiform appendix. I have noted three reports of appendicitis due to it. This would be a locality where a germ would lie longest undisturbed. I wish to draw attention to the rarity with which not only actinomycosis but tuberculosis seems to have its origin from œsophageal absorption, which can scarcely be accounted for in any other way than by the rapidity with which the infecting material passes the surface. The same may be said in regard to nasal infection. The constant drip of secretions from the upper regions of the nose tends to wash rapidly away any germs deposited in the lower respiratory channels of the nose. The sluggishness of the lower bowel gives an opportunity for the absorption of bacterial ptomaines and of the bacteria themselves, which is not presented by the upper digestive tract, due doubtless in large measure to its active peristalsis. Within the tissues we have an instance of the opportunity which stasis offers the tubercle bacillus in the preponderance of primary apical lesions of the lungs over that of other pulmonary localities.

From the consideration of these *a priori* reasons, supported to some extent by more direct evidence, the inference has been that the faucial tonsil, standing at the very forks where the breathway and foodway meet, existing normally in the form of folds and pits, but these pockets exaggerated enormously in hypertrophic disease, is the spot par excellence whence all sorts of pathogenic bacteria would start on their maleficent journey through the blood and lymph channels of the human body. From this alone we might expect to find there protective influences which prevent the annihilation of the race. More than forty years ago Virchow declared it to be

[1] Centralbl. f. Bakt., 1895, 2, p. 181.

[2] This suggestion is supported by the description of the post-mortem findings in a case reported by Lesin (Centralbl. f. Laryng., 1895, No. 11, p. 901). It is probable, as I have said, that in this case the port of entry was the tonsil, since the primary lesion was in this vicinity. There was much infiltration around a large bloodvessel in the neck, and the case terminated fatally from cerebral involvement.

entirely incomprehensible why the tonsils possess immunity in spite of the fact that they are lymphatic organs. We have learned much since then, but in spite of the horde of names recently poured into the already gorged channels of pathology by the recent researches in immunity we all recognize that we know comparatively little of protective influences. We only know they are multitudinous, and gathering them together into one word, "immunity," is convenient, but it has been very misleading. I think Figs. 3 and 4 show that the squamous epithelium lining the crypts of the tonsils is one of these protective agents against the invasion of the actinomyces. Now I have hinted that forces which keep the bacteria on the move, dripping secretions, waving cilia, intestinal peristalsis, gravity, must also be placed in the category of the defenders of the animal organism against the invasion of the pathogenic germs, these being more important in the production of that relative local immunity to which Virchow so often referred. That this keeping the germs on the move plays no inconsiderable part in slowly growing germs like the actinomyces and the tubercle bacillus, preventing them from penetrating the epithelium, we may readily believe if we are warranted, as seems the case with the actinomyces, in thinking that their toxins play a part in opening a port to their intrusion.

But if a tooth cavity will lodge a slow-growing germ long enough why will not a tonsillar crypt? And if so, why does not actinomycosis of the tonsil more frequently occur?[1]

One reason is doubtless, as we have seen, the resistance of the epithelium. Such protection does not obtain in a carious tooth cavity. There are other protective influences which are associated with the tonsil, and not with dental caries; these are gravity, mobility, exosmosis.

In speaking above of the drip of secretions rendering the upper meatuses of the nasal cavity relatively aseptic and the inferior meatus less foul than we would expect, I have intended to introduce the consideration of a factor which must obtain on all moist surfaces of the airway and foodway to some extent. The excretion of fluids, osmosis from within outward, whether through glandular or surface epithelium, must be a process which tends to wash out or keep out the pervasive germ. In the absence of the waving cilia of the epithelium, which works against gravity only in the dependent cavities, such as the accessory nasal sinuses and the pulmonary tract, gravity carries the secretions and their bacterial contents downward. These secretions themselves are apparently to some extent inimical to the existence or rather to the continued virulence

[1] The suggestion of Westenhoeffer (Berliner klin. Wochenschrift, 1904, No. 7) that the most frequent port of entry of the tubercle bacillus in infancy is through the break in the epithelium of the gums at the time of dentition, does not suffice in accounting for the usual route of entry, yet both in this breach and in the cavities of carious teeth the bacillus tuberculosis may find that opportunity which seems to furnish the most frequent access to the actinomyces.

of pathogenic germs, but the gastric secretions annihilate many of the bacteria carried into the stomach. In addition to exosmosis, which, since it has not been demonstrated for the tonsillar crypts, we may for the moment ignore, the motion imparted to the tonsillar structure by muscular action in deglutition, as in intestinal peristalsis, is sufficiently evident. The unskilled examiner is fully aware how, when he nervously forces down the tongue, the large tonsillar crypts gape in the reflex gagging of the patient. In the act of deglutition the passage of fluids and masticated solids across the mouths of gaping tonsillar follicles must tend to suck and siphon out the contents of the crypts and lacunæ. It is true of course that the configuration of some of these is such that they are thus incompletely emptied, but the conception that these cavities are the constant and crowded lurking-places of all sorts of bacterial life has been very much exaggerated. I have stained many hundred sections of tonsils to demonstrate in them the presence of bacteria. Not only have I failed in all but an insignificant number of cases to detect any bacterial forms beneath the deeper layers of the epithelium, but I have been much more puzzled to account for the comparatively few crypts which show any considerable number of bacteria outside of the epithelial wall. In very sparse numbers, it is true, with diligent search, they may be found if properly stained. Here and there one or possibly two and three cocci may be seen lodged in a nook under the upturned edge of an epithelial cell. This is the general rule, but it occasionally happens that vast numbers may be seen in a crypt, in striking contrast to neighboring lacunæ. I can only conclude that the complete or partial closure or the valve-like action of the mouth of the crypt has prevented the cleansing of the cavity in the manner I have described. These are the crypts recognized clinically by the white, cheesy masses in them, but they are insignificant in number when compared to the total number of recesses. The mechanism which keeps the latter free of visible solid matter is also a very efficient scavenger of bacteria.

Nevertheless there are enough germs in contact with the epithelium for a sufficient length of time to force ingress were they endowed with powers of penetration, but usually these bacteria are either non-pathogenic, or if they are pyogenic cocci they are non-virulent toward their host, as long at least as the latter's systemic repelling forces are in proper order. In such a swarm of microphytes as I have just alluded to a slow-growing tubercle bacillus or an actinomyces would ordinarily have an unsuccessful struggle for existence. We are fully warranted in believing that thus non-pathogenic parasitic bacteria exert at the so-called portals of infection a protective influence over the animal organism, a presumption which under other phases is a familiar and well-accepted belief.

But why is it that under such conditions of abundant bacterial life in a crypt—the cavity fairly gorged with microphytes—we can

detect none beneath the epithelium? We are familiar with the experiments of Goodale and Hendelsohn which have shown that minute carmine particles injected into the crypts or even simply blown on the surface readily pass through the epithelial barriers. Pirera[1] performed similar but not very convincing experiments with the bacillus prodigiosus, but his results are so diametrically opposite to what we observe in actual occurrence of plugged crypts that I am not disposed to accept his conclusions that bacteria also pass through the epithelium.

It is true that in acute inflammation staphylococci, streptococci, diphtheria bacilli are to be seen in the subepithelial structure. It is exceedingly doubtful if at such times we can suppose that other slow-growing forms (*e. g.*, tubercle bacilli) survive in the struggle for existence in the crypts and live to enter the tissues. In the quiescent tonsil the picture is quite a different one, and if we are to suppose that it is then the tubercle bacillus enters, it evidently differs from the other bacterial contents of the crypt.

Distrusting experiments in which a mass of powdered substance in exceptional fashion was deposited in the crypts and blown on the surface of tonsils, I studied numerous unstained sections. I sent to the dusty city of Pittsburg, and through the great kindness of Dr. Day and Dr. Jackson I was able to secure the tonsils of some of its citizens, exposed for years to the deposit of coal-dust particles. There can be no doubt that dust thus deposited in the usual way follows the same course as that deposited in the experiments of Goodale and Hendelsohn. It seems therefore that the dust passes, but cocci and bacilli of the same or smaller dimensions do not pass into the quiescent tonsil. At least if they pass they do so in such greatly reduced numbers that it leaves the contrast to the behavior of dust particles a striking and unexplained phenomenon. There are certain cases which are very rare, but which we nevertheless occasionally see in the clinics, in which are to be observed fine white points scattered throughout the tonsil itself, which are not plugged crypts. Microscopic examination will frequently show these to be tiny abscesses of the lymphoid tissue, small cavities gorged with cocci. Further examination shows that numerous cocci may be seen elsewhere in the lymph channels of the tonsil at a considerable distance from these foci.

Without pain or fever or constitutional disturbance of any kind it is evident that something has occurred here which has permitted the entrance of the surface bacteria. The cases I have seen have been in children with a phthisical family history, and the thought suggested itself to me that these individuals may also absorb dust more readily than the numerous other cases I have studied in which I made no selection. I was fortunate after some time to receive

[1] Archivii Italiani di Laryngologia, Fasc. 2, 1900.

from Pittsburg tonsils cut from children with a known marked tuberculous family history and from cases of phthisis pulmonalis. Neither in these nor in the lymphoid abscess cases could I make out any greater amount of dust passing.

We are free to infer that nature in the process of repelling the invasion of morbific germs has in the process of evolution accumulated around these diverticula of the tonsils, as elsewhere in the alimentary tube, those cells of the organism which supply the antibodies to the tissue juices, and that they are held in place by the mesh of lymph channels which may be too small at places to transmit some germs (*e. g.*, the actinomyces). This at present is a familiar conception, but from the study of the problem as indicated above we seem to be also in the presence of some wonderful mechanism of nature, evolved through æons of time, and established at the portals of infection, by which harmelss atoms glide through the stomata of cells, or their interstices, which close at the approach of the microbic enemy. When that mechanism is in perfect working order we may conjecture that the toxin of the actinomyces, the tuberculin of its more deadly cousin, is powerless to overcome it. These germs may enter immediately the undefended portals of the "predisposed," or they may lurk at the door until, in an unguarded moment—at the moment of exhaustion from illness, starvation, or other systemic disturbance—the toxin opens the path and the microphyte passes the first barrier. I do not know what part, if any, the epithelium of the tonsils may play in the production of antibacterial ferments. It is supposed by some observers that the intestinal epithelium is an important tissue in its manufacture. If so, the problem in the crypts of the tonsils may also be a chemical one, but it seems to me the attention of observers has been too intently fixed upon what follows after the microbe has passed the epithelial barrier. It is the error we are constantly falling into, the exaggeration of a single factor in the body's fight against disease. We forget that the etiology of disease is a complex problem.

I do not know how much resistance is offered by the normal epithelium to the tubercle bacillus. I have definitely shown that under morbid conditions it may pass freely through squamous epithelium.[1] You will see by a glance at Figs. 3 and 4 how the tonsillar epithelium reacts to the presence of the adjacent actinomyces. I am sure you will be struck by the resemblance to the epithelium around ulcers, especially of a syphilitic or tuberculous nature. Now after the actinomyces has passed at some unguarded gate you see how its toxin has fragmented the nuclei of the lymph cells in Fig. 5, and again we are reminded of the specific granulomata.

[1] See the plate attached to the paper on "Tuberculous Infection of the Lymphoid Tissue in the Pharynx, with some remarks on Laryngeal Infection," New York Medical Journal, September 26, 1896, read that year before the American Laryngological Association.

There is another curious analogy to syphilis at least. Van Niessen's[1] observation of the occurrence of a microphyte resembling the actinomyces in cases of syphilis has, so far as I know, received no support, but not only has the iodide of sodium a very favorable effect upon the lesions of actinomycosis, but solutions of comparatively weak strength ($\frac{1}{8}$ per cent.) retard and stronger ($\frac{1}{2}$ per cent.) solutions inhibit the growth of the actinomyces.

Whatever may subsequently be proven to be the virus of syphilis, we know the effect on cells under certain conditions of the virus of actinomyces and of the tubercle bacillus. What we have seen and what we are familiar with is the result of a destructive agent upon cell life. We know very much less accurately the biological processes which the toxin of these bacteria excite in the activity of the cells. The tuberculin evidently profoundly modifies their function. What it does in a chemical or biological way is shadowed forth in some of the terms in the recent observations on immunity, but how it affects them mechanically is entirely unknown. How the endothelium of the lymph channels may dilate or contract or become swollen we do not know, and yet that lies at the root of the problem of their carriage through the tissues. In this connection a recent remark of von Behring is suggestive:[2] "To scrofula I ascribe the alteration in the muscular coats of the blood-vessels brought about by T. B. infection, which finds an expression in excessive sensitiveness toward tuberculin, and which in general may be compared to the scrofulous diathesis of the older writers."

To pursue this subject farther would lead us too far from the portals of infection, which was the site of the activities in this unique case of actinomycosis of the tonsil.

REPORT OF A CASE OF RUPTURE OF AN ANEURYSM OF THE AORTA INTO THE LUNG, WITH SPONTANEOUS RECOVERY.

By N. S. Ferry, M.D.,
of Memphis, Tenn.

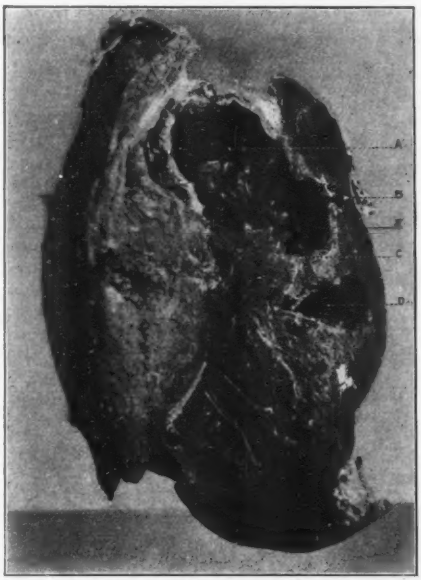
The following rare case occurred in the service of Dr. A. R. Defendorf, pathologist and assistant physician to the Connecticut Hospital for the Insane, with whom the writer had the pleasure of working during the summers of 1900 and 1901. The history of the case was kindly furnished by Dr. Defendorf.

The patient was a man of unknown personal history, except that he had had syphilis, suffered from organic dementia, the onset of which occurred at forty years of age, immediately following head

[1] Berl. klin. Woch., 1904, No. 4.

[2] Centralbl. f. Bakt., 1898, No. 2, Bd. xxiii.

injury. He was admitted to the Connecticut Hospital for the Insane three months later in a condition of pronounced excitement, which continued this time for two years. He was always in excellent physical health, and from forty-two years was successfully employed in the hospital, as he presented but a moderate degree of deterioration. At fifty-eight years (March 26, 1898) he suffered from a very profuse pulmonary hemorrhage, from which he rallied rapidly, but from this time, although physically robust and never complaining of ill-health, he discontinued work and spent his time lounging. At sixty years, February 17, 1900, without previous evident change, he was found dead in bed surrounded by a large pool of blood.



A. Upper. B. Lower. C. Point of entrance. D. Abscesses of wall.

The following is a description of the specimen in the laboratory of the Connecticut Hospital for the Insane:

The superior lobe of the left lung into which the rupture occurred is very dark in color and very firm to the touch, as if completely consolidated. On section the remainder of the lungs are found to be healthy. No signs of tuberculous or any other cavity formation.

The upper half of the superior lobe of the left lung is taken up by the aneurysmal sac "A," and is somewhat elongated, measuring 8 x 8 x 5 cm. As the lung was sectioned it appeared to occupy the space of about one-half of the lobe. At the lower end of the sac

there seems to be a dilatation forming a second sac, "E," measuring 3 x 3 x 2.5 cm. Immediately beneath this sac "E" is seen a cavity in the lung tissue "D," 4 x 3 x 1.5 cm. in size. This cavity "D" is situated in about the centre of the lobe, 9 cm. from the superior surface of the lobe and 1 cm. below the inferior wall of the aneurysm. The walls of this cavity are composed of healthy lung tissue, are very ragged, and give the appearance of having been torn apart but a short time previous. This cavity, which was discovered distended with clotted blood at the time of the autopsy, is found to communicate with the second sac "E" of the aneurysm by means of a small opening "C," through which the blunt end of a dissecting probe may be passed. The walls of the aneurysm proper are very much thickened and studded with necrotic areas varying in size from a small pea to that of a ten-cent piece, many of which are undergoing ulceration. The ulcers of the second sac "E" are very much thinner and do not contain necrotic areas similar to those found in the walls of the aneurysm itself. On the surface of the lung just over the second sac is seen a depression, "B," about 2.5 cm. in length and 1 cm. in width. On section at this point the tissue is found to be about 2 mm. in thickness. It is clearly seen that the sac approaches nearer the surface at this point than at any other. The tissue at this point is very hard, dense, and of a light color, and is composed mostly of scar tissue. It is attached directly to the pleura and appears to be the scar of some previous rupture. This theory is further borne out by the fact that the wall of the sac at the point "B" for a space of 5 cm. in distance is missing and its place is taken by the previously mentioned scar tissue. The first hemorrhage, which took place two years before the fatal hemorrhage, probably occurred through the rupture, and from some unknown cause healed of its own accord.

Beyond the lungs the aneurysm, which occupies the entire aorta for about 10 cm. in length, is fusiform in shape. The walls here are also studded with numerous areas of necrosis similar to those found in the sac within the lung.

After a fairly careful search through the literature, the rarity of this occurrence was made very apparent, as only a few cases were found where a patient had lived any length of time after a rupture of an aneurysm of the arota, and still more rare are those cases where the scar of the rupture was found. The eight cases found in the literature are of enough interest, I think, to warrant the publication of a short résumé of each.

Case I. (M. Howard Fussell, *Transactions of the College of Physicians*, Philadelphia, 1891, third series, vol. xiii. pp. 127–132. "Aneurysm of Aorta, with Healed Rupture and Recent Rupture.")—Patient, aged forty-six years, presented himself at the Medical Dispensary of the University of Pennsylvania, July 29, 1887. For two years under the care of Dr. George Dock. Complained of pain in left chest and back lasting for several months. Was put on a mix-

ture containing potassium iodide. On April 5, 1888, patient awoke at 3 o'clock with a salty taste in his mouth. Cough came on and he spat up about one pint of pure blood, frothy at first and afterward clotted. Was kept in bed and put on low diet. A note on the 30th: Sound, dark-clotted blood spat up yesterday with epistaxis.

August 19, 1891. Patient died suddenly after going to bed last night.

Autopsy Twenty-four Hours Post-mortem. On opening the cavity of the chest a considerable amount of serum exuded from the left side. All the organs were displaced to the right. Left lung was floated upward. Cavity filled with serum and blood. Aneurysmal sac began at the middle of the arch and continued to the bottom of it. The left lung was adherent to the sac, over an area of five by five inches. The adhesions were old and firm, and the lung was spread out in a thin layer over the adherent portion of the sac. The sac was empty. In some spots there were small recent clots, and one of these in the anterior part of the sac, where the lung was adherent, filled a round cavity, which proved to be a healed ulcer, the base of which was the lung. Farther back was another healed ulcer, which was half an inch in diameter. It was shallower than the other, but showed lung tissue at the bottom. One inch from the lower end of the sac, on its concavity, was a recent laceration one and one-half inches long, with irregular edges and somewhat S-shaped. This was the recent rupture which caused death.

The author remarked that the case was remarkable for the severe hemorrhage which did not prove fatal. This hemorrhage doubtless took place through the small ulcers over the adherent lung. The blood that was coughed up was frothy, and that lung showed changes after the hemorrhage.

Case II. (Wilks, *Transactions of the Pathological Society of London*, 1861, vol. xii. p. 71. "Aneurysm of the Aorta; Rupture into the Pericardium; Subsequent Closure by Inflammation.")—Male, aged sixty-two years, was admitted into Guy's Hospital under the care of Dr. Wilks, on December 6, 1860, suffering from all the symptoms of heart disease. There was considerable dyspnœa, slight yellowness of the skin, and dropsy of the legs. On listening to the chest a very slight and scarcely perceptible systolic bruit was heard over the neighborhood of the valve and limited to that spot; thus the nature of the cardiac lesion was not at first apparent. On the next visit two days afterward the man was found to have acute pericarditis, denoted by a loud to-and-fro rub over the base of the heart. The pericardial signs continued for some days, when they disappeared, giving place to the very indistinct systolic murmur at first heard. The original symptoms had increased and continued to grow gradually worse until death, January 6, 1861.

The pericardium on being incised was found to be closely adherent to the heart, but it was easily stripped off, showing that the inflam-

mation was recent; in fact, about three weeks old. At the base of the heart a small space was seen between the serous membranes, and in this was contained some clotted blood mixed with lymph. Here, also, the ascending aorta was seen bulging outward in the form of a small aneurysm, and in the centre of the aneurysm was a rent holding coagulum. "It was thus clear that this rupture had been the cause of the pericarditis which took place two days after the man's admission, and that the lymph then thrown out had prevented a further effusion of blood and a fatal hemorrhage." The aneurysm itself was about the size of a small egg, was situated in the ascending aorta, and projected forward so as to cause pressure on the pulmonary artery, which was the cause of the heart symptoms.

CASE III. (Felix Semon, *Transactions of the Pathological Society of London*, 1876–1877, vol. xxvii. pp. 76–85. "A Case of Aneurysm of the Thoracic Aorta, with Repeated Perforations in the Œsophagus.")—Patient, aged forty years, male, came on October 31, 1876, into the hospital for disease of the throat. Complained of dyspnœa, which increased at every moment, and of temporary pains in the chest, which seemed to originate under the sternum at the height of about the fourth to the sixth vertebra, and to move from there to the back. Food seemed to remain for some time at the same height, at the place of said pain, before entering the stomach. Two months before his admission he had once had a fainting fit without any known reason, since which time the stoppage of food is said to have begun. The diagnosis was made of "aneurysm of the arch of the aorta of a considerable size, but nowhere reaching the chest wall." Patient began to grow worse, vomiting set in, and during the last few days a short, dry, distressing cough began.

Post-mortem Examination Made by Dr. Barrett. The arch of the aorta from the very beginning to the middle of the descending part was considerably enlarged. At the height of the fourth to the sixth vertebra was a nearly globular aneurysm of the size of a small orange. It was united to the œsophagus and vertebral column.

The wall of the aorta was much thinner than normal over the entire extent of the adhesions to the œsophagus. The anterior wall of the latter was perforated circularly at the height of the fifth vertebra. The opening was about the size of a silver dollar. In this opening appeared a thoroughly organized, conical, brownish-red thrombus of the size and shape of the first phalanx of the little finger, which originated from the uppermost part of the posterior wall of the aorta. Its surface is rough and torn. In the middle of the posterior wall of the aneurysm was found a recent small perforation in the œsophagus. The author thought that the presence of the large thrombus in the œsophagus proved that the first bleeding must have taken place some time previously, probably when the patient had his fainting fit, three months before admission, and from which he also dated the difficulty of swallowing. The trickling of

blood must have occurred by drops and through a small opening, so that the extravasated blood had time to coagulate and to prevent, by the formation of coagulations around the perforations, an enlargement of the latter.

CASE IV. (Case reported by Dr. C. Hubner at the Hospital San Juan de Dios, at Ramanga, Chili.)—L. A., aged fifty-two years, admitted to the hospital April 9, 1862, on account of asthma trouble. Patient appeared to be slightly dropsical. A short time before admission he had vomited about four pounds (pints) of blood without much coughing. Color of blood was pale red. I diagnosed case "hypertrophy of heart and aneurysm of aorta." He remained in the hospital a few weeks and was discharged on his own request.

On February 12, 1863, he was again admitted for the same trouble. Had vomited a large quantity of blood about fourteen days previous. The hemorrhage had entirely ceased after about thirty hours. He was suffering greatly from asthmatic trouble and had severe fits of choking. I again diagnosed case "aneurysm of aorta." Under treatment he improved, and on February 26th asked to be discharged, which was refused. On February 27th he suddenly began to cough, spat up about two pounds of blood, and died within ten minutes.

Autopsy. Aneurysm found over the upper portion of the left lung filled with a mass of clotted blood. Lung tuberculous. The autopsy led me to believe that the aneurysm had existed for a long time and had ruptured twice. When he threw up the large amount of blood the first time a rupture had occurred; when hemorrhage suddenly stopped the blood became clotted in a layer over the rupture, forming a pseudomembrane. We thus have a spontaneous healing of a rupture of an aneurysm of the aorta. The second rupture, which was quite open and not covered with clotted blood, was the cause of death. There was extensive necrosis and loss of the periosteum of the spine, caused by the continued and permanent pressure of the aneurysmal sac on the spine.

CASE V. (Surgeon F. R. Town, United States Army, Fort Sill, Indian Territory. *Reports of the Surgeon-General of the Army*, Washington, 1885, 1886, 1898. "Report of a Case of Double Rupture of an Aneurysm of the Ascending Aorta, with Survival of the Patient for Several Weeks Subsequent to the Primary Rupture; Final Result, Death.")—Patient, aged thirty-two years, Mexican by birth, appeared at sick call complaining of shortness of breath and inability to blow his instrument, a brass horn. Four days afterward he was seized in quarters with a sudden faintness and alarming prostration. He was seen immediately by Acting Assistant Surgeon J. L. Powell, who found an entire cessation of respiratory movement, a murmur over the right side of the thorax, and universal flatness on percussion, while the respiratory movements of the ribs on the left side were evidently increased. Marked pallor of the face and

skin prevailed and the pulse was extremely feeble. Patient improved until May 28th, when, as he was in the act of sitting down to dinner, sudden faintness came on and he vomited a large quantity of fresh arterial blood. At 9.30 P.M. a violent hemorrhage occurred from the mouth and nose, with an almost immediate fatal result.

Autopsy Thirteen Hours after Death. The right pleural cavity contained a large amount of disintegrated coagula and fluid blood, which had evidently extravasated some time prior to death. Considerable damage had resulted to the superficial parenchyma of the lung posteriorly, especially near its posterior border, through which the escaping fluid had effected an entrance from the descending aorta. The pleural surfaces were firmly adherent to the chest wall and also to the lung itself, along the external surface and base. These adhesions were old and had limited the amount of extravasation. "To their strength was probably due the temporary preservation of the patient's life by preventing the general diffusion of the escaping fluid throughout the pleural sac and the forcible compression of the lung into the apex of the thorax." About one inch and a quarter below the origin of the left subclavian artery a sacculated aneurysm of moderate size, with a large mouth, projected from the right of the descending aorta in front of the œsophagus. The aneurysmal pouch was oval, and from one to two inches in diameter. A circular ulceration through the sac, about three lines in diameter and immediately over the œsophagus, indicated the position of the fatal rupture. After a little search a second small opening through the sac nearer its mouth was found, which communicated to the right and behind the œsophagus directly with the extravasation within the right pleura. The sac contained a small amount of clot and was partially lined with a thin lamina of fibrin, which was believed to have occluded the passage to the right thorax.

CASE VI. (Theophilus Thompson, *London Medical Gazette*, 1840, vol. ii. p. 905. "A Case of Aneurysm Supposed to Have Opened into the Pericardium a Considerable Time Before Death.") —On July 21, 1839, the patient, a blacksmith, of strong, muscular frame, was found lying in bed, lips and ears slightly livid, skin perspiring, and countenance rather anxious. Complained of pain on the right side of the sternum, increased by full respiration. Right chest was prominent, especially over the third, fourth, and fifth ribs, where the sound elicited by percussion was dull, while the natural dulness of the cardiac region to the left of the sternum did not extend so far as usual. Physician concluded that the heart was enlarged and the aorta studded with ossified depositions." He could not satisfy himself whether there was aneurysm of that vessel. Twelve months previously the patient had been attacked with pain in the back and side, since which time he had suffered from palpitation, and had occasionally fainted at his work. On the following

September the patient became worse, and on the 19th he was found again confined to bed with great lividity of countenance and a very weak and rapid pulse. He was treated at that time and improved until the 29th, when, on getting up for stool, he became suddenly worse, stared wildly for a moment, uttered an exclamation, and fell back dead.

Post-mortem Examination. Pericardial sac filled with clot. Heart enlarged and flaccid. Aorta near its origin was dilated to more than twice its ordinary size. Inner membrane seemed to have lost its natural elasticity. No ossification. At the most dilated part of the aorta an opening was discovered, the edges of which were even as if cut with a knife. This was probably made in removing the heart, but at one end of the wound, two or three inches above the aortic valve, was a somewhat puckered opening, apparently produced by disease. On tracing this aperture it was found to communicate with a sac. This pouch, which was larger than an orange, was full of dense coagulum and lined with fibrinous layers. At its outer side, nearly on a level with the aortic valves, was a round smooth aperture, rather more than three-quarters of an inch in diameter, through which the blood appeared to have escaped into the cavity of the pericardium.

"It seems reasonable to conclude that in this case the pericardium had not only been thickened by inflammation at some distant period, but had also been affected with more recent inflammation up to the time of death."

The indication of great displacement of the heart at an earlier period, the roundness and smoothness of the opening into the pericardium, the great distention of that membrane, the increased firmness of its structure, the evidences of recent and progressive inflammation seem favorable to the opinion that the rupture of the aneurysm into the cavity of the pericardium had occurred a considerable time before the death of the patient.

Case VII. (Charles Murchison, "Diffuse Aneurysm of the Thoracic and Abdominal Aorta, Terminating in Chronic Peritonitis, with Copious Liquid Effusion.")—Patient, James D., aged forty-two years, on January 1, 1869, suddenly felt ill and then immediately became unconscious, remaining in this condition for an hour. Following this at the beginning of August the patient had a severe attack of pleurisy, without any strain or unusual exertion was suddenly taken with urgent vomiting, severe pain, and distention of abdomen and constipation. On September 4th the sickness recommenced and was attended by pain in the stomach, but less severe than on former occasions. On the 10th he became worse, and since then had great pain and vomited everything he swallowed. His bowels had been relaxed and shortly before his admission had passed a considerable quantity of semicoagulated blood. He gradually became worse, and died on the morning of the 23d. At the

post-mortem an aneurysm was found at the lower part of the thoracic and upper part of the abdominal aorta.

COMMENTS BY MURCHISON. On January 1st the aneurysm ruptured and caused syncope. On August 4th the aneurysm burst in a downward direction and, the blood pressing forward, the liver and the peritoneum caused chronic peritonitis.

CASE VIII. (Dr. Rhodes, *London Medical Times and Gazette*, 1863, vol. ii. p. 10. "Rupture of an Aneurysm into the Left Pleural Cavity; Death Three Days after the Supposed Time of Rupture.") —T. W., aged thirty-nine years, decorator, was admitted into St. Mary's Hospital on February 11th, with supposed effusion into left lung. One week ago he became suddenly faint and fell down insensible, in which state he remained two minutes. On recovery he felt considerable pain on the left side. Was supposed to have received some injury to ribs. On examination the whole of left side was found dull on percussion, but absence of breath sounds and vocal vibration. No pulsation in cardiac region, but the apex beat could be felt in the epigastrium and the heart sounds to the right of the xiphoid. Death on the 14th.

Autopsy. Left pleura was filled with dark-colored serum. Heart displaced. Large clot of blood measuring four pints was effused over surface of the lung. On removing this a sacculated aneurysm about the size of a small orange was found at the posterior part of the thoracic aorta just at its termination, which communicated with the pleura by an opening about the size of a quill. No trace of pleurisy found.

If any one situation is more favorable than another for the spontaneous recovery after the rupture of an aneurysm of the aorta, the lung seems to enjoy that distinction, probably accounted for by the better facilities offered for the rapid formation of a clot with which the rupture is blocked. A number of cases are recorded where the patient lived a few hours or perhaps a day or two after the rupture of an aneurysm into the lungs, pleural cavity, or pericardial sac, but these cannot be placed under the heads of recoveries.

PRIMARY TUBERCULOSIS OF THE BREAST.*

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WHEN the function of the breast is taken into account, one would expect mammary tuberculosis to be as frequent as genital tuberculosis. This is certainly not true. Among 300 gynecological cases

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Stratz[11] found 22 per cent. of tuberculosis; Martin had 10 in 287 cases, and Whitridge Williams reported that 8 per cent. of inflammatory adnexal disease was due to the tubercle bacillus.

Mammary tuberculosis is more rare. Bartsch[9] in 1901 could find but 65 undisputed cases in the literature. Probably 30 of these were primary. The primary form of genital tuberculosis has been open to doubt. Such pathologists as Klebs, Scanzoni,[11] Bollinger, von Recklinghausen, Schmorl, Ribbert, Aschoff and Albrecht[12] deny its existence in the adult. It is as difficult to prove the actual primarity of tuberculosis of the breast as it is of the genital organs. This does not apply to cases of direct inoculation, for well-attested instances of the latter are on record. Thus, Pluyette[1] reports primary tuberculosis of the breast in a girl aged twenty-three years, who was infected through a slight abrasion of the nipple, and Spinelli[13] speaks of a woman who developed the disease after intercourse with a male who suffered from tuberculosis of the testicle.

Whenever the infection is conveyed by means of the circulatory systems the question always arises whether, if autopsy were possible, the primary focus of the disease would not be found in the visceral organs more often affected by tuberculosis. Without an autopsy, therefore, a diagnosis of primary tuberculosis is not scientifically justified.

Primary genital tuberculosis does occur, however, and Veit,[22] after scrutinizing all of the available cases, accepts 15, 10 of the tubes and 5 of the uterus, as actual primary forms.

There are no cases of primary tuberculosis of the breast on record in which the diagnosis was made after autopsy. But as the primary form occurs in the genitalia, it is reasonable to suppose that it may develop in the breast. It is certain that tubercle bacilli may produce characteristic lesions remote from their point of ingress without affecting the parts through which they pass. Dobroklonski,[18] working under Cornil, and Ravenel[18] have produced experimentally primary tuberculosis of the lungs through the alimentary tract without any lesion of the tract itself. Attention has recently been directed to the tonsils as frequent portals of entry for the tubercle bacillus. That tubercle bacilli may be present in the tonsil without producing any anatomical alteration is evident from the work of Friedmann.[18]

Goodale and Hendelsohn[14] have observed that the tonsils possess marked absorptive powers. The crypts of the tonsils represent afferent lymph vessels, and carmine granules placed on the tonsils are rapidly absorbed and pass thence through the interfollicular tissue to the efferent lymphatics. As these facts might explain the occurrence of primary tuberculosis anywhere in the economy, we may accept, for clinical purposes at least, those cases of tuberculosis of the breast as primary in which there is no evidence of tuberculosis elsewhere, even after the most careful physical examination.

The importance of primary tuberculosis of the breast lies in the possibility of its transmission from the mother to the nursing child.

While it would be quite unusual, at least in the better classes, for a woman with any disease of the breast to suckle her infant, yet Coles[7] saw, in the out-patient department of the Philadelphia Polyclinic, two women with tuberculous breasts nursing tuberculous infants, and Davis[7] believes that he has made the same observation at the General Hospital in Vienna.

So far as I know there is but one case in which tuberculous infection of the child has been traced to its mother's milk. This case, reported by Roger and Garnier,[10] may be briefly summarized as follows:

A pregnant woman came under observation on February 16th, suffering with pharyngeal tuberculosis. On March 7th she gave birth to a child that weighed 2685 grams. The infant was nursed until March 12th, when, on account of the progressing illness of the mother, it was weaned. On March 24th the woman died, and at the autopsy tuberculosis of the lungs and other viscera was noted. The child gradually failed and died on April 25th from tuberculosis of the mesenteric glands, spleen, and kidneys. Two guinea-pigs were inoculated with the milk of the mother on March 11th. Both of these pigs died, one with tuberculosis, the other with fibrous peritonitis and enlarged lymph glands; no tubercle bacilli nor actual tubercles were found in the second case, but the lesions were evidently due to the tubercle bacillus.

While such an observation is exceptional in woman, it has been frequently made in the cow. Pearson[17] quotes many instances of infection arising directly from the milk of tuberculous cattle. Ernst found tubercle bacilli in the milk of 33 per cent. of cows in which a complete post-mortem showed that while the animals were widely tuberculous the udder was free; and Hirschberger, by the experimental inoculation of milk, produced tuberculosis in 55 per cent. of cases from tuberculous cows whose udders showed no sign of disease.

We may therefore believe that the milk of a tuberculous woman is always dangerous for the child, and that, although rare, tuberculosis of the breast may occur in a primary form and be unsuspected, and that the possibility of this condition ought always to be borne in mind during the period of lactation. If the breast is itself the seat of tuberculosis its milk must be highly infectious, for in the analogous affection of the cow the milk almost invariably contains tubercle bacilli.

There is some difficulty in reaching a satisfactory estimate of the frequency of primary tuberculosis of the breast. Freiburg[6] mentions 50 cases, Scudder[8] 80, Carrel 120. Bartsch[9] has thrown out of consideration all those in which a positive diagnosis was not made. Thus in all of his cases, either there was the histological picture of tuberculosis or the bacilli were found or inoculation experiments were positive. There were but 65 such cases, and this is the number

Bartsch includes in his series. So much for the existence of a tuberculous lesion.

When we attempt to separate the primary from the secondary forms there is difficulty also in determining which are primary. While it is unscientific to diagnose primary tuberculosis of any part without an autopsy, we may safely consider a given lesion primary if there is no evidence of tuberculosis elsewhere. If in a given case symptoms exist which may be attributed to tuberculosis in another part, then the disease of the breast cannot be regarded as primary, although the breast may have been its primary seat. This is the opinion of Piskacek,[16] and with it there are no grounds for disagreement.

Tuberculosis of the breast as a secondary lesion has no especial significance, whereas the fact that the mammary gland alone may be affected by the tubercle bacillus is of practical importance. It is well recognized, of course, that in the later stages other organs may be involved.

Reviewing Bartsch's series, in 35 there seems to have been tuberculosis elsewhere. All cases designated as being of a scrofulous habit and those in which there was coincident but less-marked tuberculous infection have been excluded. I have also excluded those cases where the affection was first noted in the axilla. Many of the tabulated cases are incompletely reported and details for determining the actual primarity of the case are scanty. At any rate, Bartsch has no more than 30 primary cases, and 2 of these are in the male. To these I have been able to add 12; some of the latter were not included by Bartsch in his list because suitable literature was not available. The impression one has in going over the reports of tuberculosis of the breast is that its primary form is extremely rare. Bull[21] in 1894 had had 185 amputations of the breast; all of these were submitted to histological examination, and in but one instance was tuberculosis found, and that was primary. Since that time Bull has not observed another case, although he has no record of the number of his breast amputations.

If we analyze the entire number of accepted cases in the female we find that of 40 patients, the entire number—

28	were married	70 per cent.
19	had borne children	47.5 "
12	were single	30 "
12	had hereditary taint	30 "
6	had history of trauma	15 "
8	suffered from mastitis during lactation	20 "
2	were directly inoculated	5 "

The youngest patient was sixteen, the oldest was fifty-nine years. From these figures it is difficult to draw many conclusions in regard to the predisposing causes of mammary tuberculosis. While most of the cases were found in married women, the age at which the disease occurs would explain this fact, because most women

between the ages of twenty and forty are married. No case up to this time has been observed in the female before the age of puberty, although Demme reports a case in a male child four days old, due to direct inoculation. There seems to be no direct connection between lactation and the disease; but 47.5 per cent. of the women had borne children. Trauma has played an inconsiderable part. Perhaps the relative infrequency of mammary compared to genital tuberculosis is to be explained upon other grounds. Hegar, Martin, and Amann[11] have dwelt upon gonorrhœa as a predisposing factor in genital tuberculosis. Eisendrath[19] has recently made the same observation and believes that the gonococcus often prepares the soil for a later invasion of the tubercle bacillus. He quotes Casper,[20] who found 33 per cent. of 35 cases of tuberculous vesiculitis to have been preceded by gonorrhœa. Casper believes that an attack of gonorrhœa, especially in those predisposed to tuberculosis, acts like a trauma in aiding its development. It is similar to an injury in syphilis which is able to produce the manifestation of the disease at the injured spot.

PATHOLOGICAL ANATOMY. Tuberculosis of the breast occurs in either a (1) confluent or a (2) disseminated form.

The confluent form may be an instance of (*a*) mixed infection or of the (*b*) true tuberculous abscess.

The confluent abscess in the presence of mixed infection is the usual variety. It may involve the entire breast; it breaks down, forming fistulæ, and in the later stages produces retraction of the nipple. There are the usual signs of inflammatory processes in this region; the axillary glands may be secondarily involved.

An abscess purely the result of the tubercle bacillus, the so-called "cold abscess," is quite rare. It forms an elastic fluctuating tumor, mostly well circumscribed and sharply bordered. It is but seldom surrounded by infiltration; the skin remains unbroken and there are no fistulæ. The axillary glands are not involved.

In the disseminated form there are small nodes of the tuberculous disease scattered throughout the breast. There is not much general enlargement of the gland; the skin remains unaltered; the disease is slow in course, years passing, perhaps, without cognizance of the patient. This form is so rare that it is denied by some authors.

DIAGNOSIS. Many tumors of the breast and the inflammatory affections have to be distinguished from tuberculosis. The differential points here would be like those of tuberculosis in other parts and need not be repeated. In suspected cases the mammary secretion should be stained for tubercle bacilli, as was successfully done by Bevan,[7] or animal inoculation should be made. From simple pyogenic mastitis tuberculosis may be, as a rule, distinguished by its more chronic course. Carcinoma usually occurs later in life and is accompanied by retraction of the nipple. This does not often result in tuberculosis except after the formation of sinuses.

Reported by	Age and social state.	Hereditary history.	Previous disease.	No. of labors.	Last labor.	Lactation.	Breast affected.	Original seat of disease.	Trauma.	Treatment.	Duration of cure.	Remarks.	Axillary glands involved.	Upon what diagnosis is based.
Pluyette,	23 years; single.	.....	.....	.....	.....	.....	.....	Breast.	.....	.....	.....	Direct inoculation.	.....	Bacilli were found in the nipple.
Salomoni,	21 years; single.	.....	Nodule in breast 2 years.	.....	.....	.....	Right.	Breast.	.....	Amputation of breast.	2 yrs.	.....	Yes.	Bacilli.
Salomoni,	23 years; married.	.....	.....	.....	.....	.....	Left.	Breast.	.....	Amputation of breast.	4 yrs.	.....	Yes.	Bacilli.
Warthin,	40 years; married.	.....	.....	5	11 mos. before.	Breast pained while nursing 3d child, 2 yrs. before last.	Right.	Breast.	....	Excision of nodes.	1 yr. ?	.....	.....	Bacilli; histological examination.
Warthin,	39 years; single.	Yes.	Blow on breast 5 yrs. bef're	.....	.....	.....	.....	Breast.	Yes.	Amputation of breast.	Not heard from.	.....	Yes.	Histological examination.
Davis,	17 years; married.	.....	.....	.....	Pregnant 7½ mos	.....	Right.	Breast.	.....	Amputation of breast.	1 yr	Allowed to go to term; baby died in 6 mos. of marasmus.	.....	Bacilli.
Davis,	25 years; single.	.....	.....	.....	.....	.....	Left.	Breast.	.....	Amputation of breast.	8 yrs.	.....	.....	Histological examination.
Powers,	40 years; married.	Yes.	Pain in rt. breast ten years before at 2d child.	6	.....	Never nursed with right breast after second child.	Right.	Breast.	.....	Excision of nodules.	3 yrs.	.....	.....	Histological examination.
Powers,	26 years; married.	.....	.....	1	2½ yrs. before.	Had not used left breast for nursing; no reason given.	Left.	Breast.	.....	Amputation of breast.	3 yrs.	Patient subsequently died of phthisis.	Yes.	Histological examination.
Ely,	35 years; married.	.....	.....	.....	.....	.....	.....	Breast.	.....	Amputation of breast.	Not given.	Direct inoculation.	.....	Histological examination and animal inoculation.
Schifone,	32 years; single.	.....	.....	.....	.....	.....	Right.	Breast.	.....	Amputation of breast.	Not given.	.....	.....	Histological examination and animal inoculation.
Anspach,	59 years; married.	.....	.....	3	17 years before.	.....	Right.	Breast.	.....	Amputation of breast.	1 yr.	.....	.....	Histological examination.

PROGNOSIS. As in genital tuberculosis, the disease in itself is favorable. The result depends upon the existence of visceral complications. Bartsch's data relative to the results of treatment in his series is too meagre to draw conclusions. In my own series of 12 cases, 2 were not heard from after operation; 4 are reported cured within a year; 1 died at the end of three years, and in 1 the after-condition of the patient is not noted. The duration of cure in the remaining cases is put down at 8, 4, 3, and 2 years.

TREATMENT. Although simple evacuation and curettage or extirpation of the diseased nodules alone has sufficed in some cases, the gravity of the disease usually demands amputation of the breast and the removal of enlarged axillary glands.

The details of the case I have added to those found in the literature are as follows:

HISTORY. Woman, aged fifty-nine years, the mother of three children; there is no tubercular history; there is no clue whatever to the probable source of infection. Husband and children are all living and well. Parents died of old age. Patient married at the age of twenty-eight; menopause at forty-eight; last child when forty-two years of age. She always used both breasts during lactation and never had pain in them. Never had any pulmonary disease nor any illness that might be construed as being tuberculous in nature. Three years before coming under observation a nodule appeared in the right breast; it increased slowly in size and caused retraction of the nipple. There was no ulceration. Pain occurred in the right arm.

Treatment. The breast was amputated by Dr. J. G. Clark, who saw the woman in consultation with Dr. James Tyson. I wish to thank these gentlemen for the privilege of reporting the case.

Pathological Report, Path. No. 607.

Macroscopic Description. The specimen consists of an amputated breast including a portion of the pectoral muscle, together with axillary fat and lymph glands. The skin surface is intact; the nipple is considerably retracted, and at this area and to the right of it there is an irregular nodule about the size of a walnut. The mass is not well circumscribed, but fuses with the surrounding tissue. Upon section the breast appears atrophic. Beneath the nipple and continuous with it there is an irregular mass made up of fibrous tissue. Here and there are dark spots which appear to be areas of necrosis. The entire glandular portion of the breast is represented by this nodule. Its fibres are continuous with the trabeculæ of the fatty tissue which makes up the bulk of the specimen.

Histological Description. The surface epithelium and that of the ducts appear normal. The glandular elements are quite atrophic and there is a relative increase of connective tissue. The lymph spaces throughout the specimen are crowded with small cells having

round nuclei which occupy most of the cell body. Such collections are directly continuous with areas comprising giant cells surrounded by epithelioid cells and the small round ones above mentioned. The nuclei of the giant cells are at the periphery, and the picture is typical of tuberculosis. A few sections were stained for tubercle bacilli, but none were found. It is unfortunate that no animal inoculations were made, but as the case was clinically one of carcinoma the specimens had been hardened in formalin before the value of such proof was apparent.

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A CASE OF MERALGIA PARÆSTHETICA.

By John E. Donley, M.D.,
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I wish to add the following case of meralgia paræsthetica to the number of those already reported:

Mrs. G., white, aged forty-three years, primipara, came under observation complaining of disability in the left thigh. Her father is living and well at the age of sixty-eight. Her mother died of heart disease at the age of fifty. She has one brother and one sister, both living. The former enjoys excellent health, the latter suffers from intercostal neuralgia. A maternal aunt died of cancer of the breast, and a paternal uncle of Bright's disease. Apart from measles and chicken-pox the patient enjoyed good health until the birth of her child nineteen years ago. The labor was difficult and the puerperium was complicated by abscess of the breast and a

mild form of mania, which persisted for three weeks. From recovery her health was good until one year ago, at which time her husband's misfortune in business occasioned an attack of neurasthenia which lasted about six weeks. She is a woman of high-strung temperament, plethoric habit, medium stature, and heavy build. There is no history or sign of alcoholism or syphilis. She uses tea and coffee moderately. There has always been a tendency to constipation. At present she is passing through the changes of the climacteric and suffers from occasional flushings, dizziness, and palpitation. Twelve weeks before seeking medical attention and while in the enjoyment of her usual health she dropped a flat-iron on her right foot, injuring it severely. For one week she was unable to walk, but gradually regained her power, the weakness of her right foot, however, compelling her to throw as much weight as possible upon the left leg. This because of her weight caused much fatigue. Eight weeks after her initial injury she began to notice a peculiar, rather ill-defined feeling of tingling and numbness accompanied by a sense of constriction on the outer side of the left thigh. Except on four occasions this sensation was entirely absent during rest, but appeared shortly after she began to walk or stand and increased to positive pain which grew progressively worse until she was obliged to sit down. These symptoms continued to increase for five weeks from their inception, when she came under observation. It was impossible for her to walk without a limp, but she had no abnormal sensations in the limb, other than those in the outer thigh. Apart from tingling, numbness, and pain she complained at times of intense heat, at others of cold, as though a wet cloth were placed upon the skin. Occasionally the outer side of the thigh felt "like a foreign body," as she expressed it. Menstruation or changes in the weather appeared to have no influence on her condition. Three weeks after the onset of her symptoms she suffered from a slight attack of supra-orbital neuralgia which lasted three days and has not re-appeared. Examination disclosed anæsthesia on the outer side of the left thigh extending in a line from the great trochanter to the head of the fibula, from the 3d to the 36th cm. mark. The area was 4 cm. broad above, 14 cm. broad in the middle, and 8 cm. broad below. Within this region pain, temperature, tactile, pressure, and hair sensations were greatly diminished, particularly those of pain and temperature. Sensibility to the faradic current was likewise markedly decreased. The nerve was not anywhere sensitive to pressure, although this point was carefully investigated several times. There were no trophic changes. The right thigh was examined for perversion of sensibility of all forms, but with the exception of a small area of doubtful tactile anæsthesia at the middle of its outer surface no alteration was found. With the idea of relieving the pain and paræsthesia the patient painted a portion of the thigh with iodine. It set up a severe reaction which was the immediate cause of her

seeking medical attention. Under the use of strychnine, massage, and galvanism her symptoms rapidly improved and in three weeks they had completely disappeared.

Meralgia paræsthetica is the name given by Roth to a peculiar syndrome appearing most commonly, though not exclusively, in the distribution of the external cutaneous femoral nerve. Meralgia paræsthetica, like most other descriptive terms, is unsatisfactory, in that it describes too little, and fails to include the objective symptoms that are such an important part of the disease. On this account Sollier has suggested the name neuralgia paræsthetica, but to this also there are objections, and it would seem better, until we obtain a term based upon a distinct pathological conception, to remain content with Roth's original description. The disease, although it had been several times previously observed and reported by Bernhardt, Pieracini, Musser and Sailer, and others, was not described as a definite morbid entity until 1895, when Bernhardt published his paper in the *Neurologisches Centralblatt.* Since then a considerable number of cases have been reported, although the affection would appear to be not common, or, if it is so, it is often unrecognized. Meralgia paræsthetica may be defined in the words of Musser and Sailer as a "disturbance of sensation on the external surface of the thigh, characterized by various forms of paræsthesia, associated with dissociation and more or less diminution of sensation." Another definition would be to call it an alteration of sensation on the outer aspect of the thigh characterized by pain, dissociation, anæsthesia, and paræsthesia.

The symptomatology of the disease is quite constant so far as the kind of symptoms is concerned, but the combinations in which they appear are very various. They manifest themselves objectively and subjectively. In a large number of cases the subjective symptoms consist of tingling and numbness, slight at first, but increasing in severity as the leg is used, sometimes amounting to a severe ache or pain. Other patients complain of heat and others of a sensation of cold, or both of these may be present at different times in the same patient, as occurred in our case, where at one time the heat was so marked that the patient declared she could almost feel it through her dress, and at another the sensation of cold was compared to a moist cloth pressed against the skin. Sometimes there is hyperæsthesia so great that the slightest touch is unbearable. Musser and Sailer mention an instance of this—the case of a distinguished jurist whose thigh was so sensitive that the very touch of his clothing became unendurable and he was several times obliged to stop walking in order to pull his garments away from his skin. In the vast majority of cases the subjective sensations disappear when the patient is sitting or lying, but sometimes they persist as in our case, where on four occasions they continued despite the fact that the patient was resting quietly in bed.

The objective symptoms are those of anæsthesia, which includes all qualities of sensation in different degrees. In our case the diminution of electrosensibility was so considerable that the patient bore with equanimity in the left thigh a faradic current which caused much discomfort in the right. There is marked diminution of sensibility to pain, touch, and pressure. Our patient presented such a degree of thermoanæsthesia that a test-tube of hot water was recognized as being only lukewarm and ice gave merely an indistinct feeling of cold. In many instances, as pointed out by Bernhardt, there is tenderness of the nerve trunk just in front of and below the anterior-superior iliac spine. This was never present in our case, and, though frequent, is not a constant symptom.

Meralgia paræsthetica is usually an affection of the external cutaneous nerve, but it may be found in the anterior crural nerve, as in the cases of Féré and Möller, or in the external popliteal nerve, as in Sollier's case.

The etiology of the disease is obscure. A variety of causes have been mentioned by different observers, but it would seem that some of them at least are merely coincidences. Thus some have mentioned the presence of constipation and a sedentary life as having an etiological significance, but these terms convey nothing that is definite and bring to our minds no satisfactory information. There are, however, three factors that appear to bear a close relation to the production of the disease; these are infections, traumatisms, and intoxications. In several instances the meralgic symptoms were consequent upon some infectious process. Bernhardt, Roth, Musser and Sailer, and others mention typhoid fever, erysipelas, and influenza. In one of Nartowski's cases the attack followed a pneumonic and in one of Claisse's a malarial infection. Syphilis or its legacy, tabes dorsalis and general paresis, has been reported by Bacelli, Venturi, Pitres, and Möbius. In one case of Bernhardt's there was chronic lead poisoning; and eclampsia, rheumatism, and alcoholism are mentioned by other writers. Traumatism bears oftentimes an apparently direct relation in the production of the disease. In our case the symptoms followed directly upon excessive use of the limb, which may be regarded as a species of traumatism. In one case of Bernhardt's and in one of Knauer's a sword striking against the leg produced the symptoms, and in another of Roth's the affection followed rough walking. The pressure of a heavy army belt caused the symptoms in both thighs in a patient of Musser and Sailer. McCarthy reports a similar case of Spiller's in which the cause of the symptoms was inexplicable until a truss was discovered pressing upon the external cutaneous nerve. A neuropathic habit seems to have some influence in the etiology. Our patient suffered previously from mania and neurasthenia, while during the persistence of her meralgia she was attacked by a slight supraorbital neuralgia. Hereditary influence has been traced by Musser and

Sailer, in one of whose cases the trouble appeared in son, father, and grandfather, and in another the same complaint affected father and daughter.

The pathology of the disease is a mooted question, some authors placing it among the neuralgias, others among the neuritides, and still others denying that it should be classed with either of these diseases. Spiller believes many of the cases are allied to the pressure palsies. Musser and Sailer are of the opinion that it is probably a neurosis. In attempting to classify meralgia paræsthetica we are confronted at the outset by two difficulties: first, our ignorance of the pathological conditions present in each case, and, second, our inadequate knowledge of the exact relations neuralgia and neuritis bear to each other. Many years ago Trousseau pointed out that neuralgia is only a symptom and connotes no constant pathological condition. Neuritis likewise is a common name for different morbid changes. We have, for example, the parenchymatous, the interstitial, and the segmental of Gombault. The two affections are not mutually exclusive. In cases of neuralgia all the various processes of neuritis with degeneration and atrophy of nerve fibres have been seen. On the other hand, many cases diagnosed clinically as neuritis are accompanied by neuralgic pain. Spiller and others have demonstrated marked pathological alterations in the Gasserian ganglion and nerve trunks in trigeminal neuralgia, and Thomas has found very similar changes in the ganglia on the posterior nerve roots in the early stage of tabes, which begins with neuralgic pains. The two conditions, neuralgia and neuritis, overlap, as it were, and perhaps we are not always logical in attempting to separate them. In the few instances in which the nerve has been examined in meralgia paræsthetica pathological changes were sometimes present, as in the cases of Nawratski and Sollier; again, they were apparently absent, as in the case of Souques. But, if we admit a priori that disturbance of function is always accompanied by alteration of structure, however slight and temporary, it would seem that, even in those cases that appear normal, some change has occurred that has escaped detection, either because it is more or less fleeting or because our technique is inadequate. Possibly it will be found that in every case of meralgia paræsthetica some structural change has occurred, and when we know the nature of this we will be able properly to classify it as neuralgia, neuritis, or neuroses.

The treatment instituted in our case was the following: one-fiftieth increasing to one-thirtieth of a grain of strychnine sulphate was given three times each day. For fifteen minutes each day the limb was massaged, and following this the whole thigh was wrapped for half an hour in hot Turkish towels. At the end of a week a mild galvanic current supplemented the above treatment. Rest for the limb was insisted upon, and in three weeks the symptoms had completely disappeared. The employment of strychnine

was suggested by Dana's use of it in trigeminal neuralgia. For a week no other treatment was employed in order that the effects of the drug might be observed. From the first the symptoms began to improve, so that strychnine would seem worthy of a trial in these cases. Electricity in the form of the dry faradic brush was first suggested and used by Roth in a number of cases. Musser and Sailer report a case cured by this means, and Sailer has effected cures in two other cases by the same method. In our case we employed a galvanic current, with the anode on the thigh and the cathode on the lumbar spine with apparently good results. When the disease was attributed to rheumatism salicylic acid was given internally, while Devic employed the drug by inunction. That counterirritation is harmful in these cases was shown unmistakably by ours, where the use of iodine caused a painful dermatitis with considerable subcutaneous œdema. Spiller was the first to suggest resection of the nerve in obstinate cases. The operation has been done by Souques and Chipault on the external cutaneous nerve and in Sollier's case on the external popliteal nerve—in each instance with the relief of the meralgic symptoms. In a case recently reported by Neisser and Pollack, Poupart's ligament was divided, with excellent results.

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SURGICAL SCARLATINA.

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Surgical scarlatina, or scarlatina which follows wounds and operations and bears a direct relation to these, has been recognized for the last forty years as a distinct disease. Although from time to time doubts as to the diagnosis of reported cases have been expressed and the suggestion made that the eruptions in question were due to other causes than scarlatinal infection, on the whole, it has been generally admitted that a wound or injury constituted a predisposing cause to scarlatinal infection, and that such infection should be looked upon as a not uncommon complication of operations or injuries. There are about 174 cases of surgical scarlatina reported in detail in the literature and passing mention is made of many more. The first description of scarlatiniform eruptions after operation was made by Civiale in 1858, but he did not connect such eruptions with scarlet fever. He states that "it is quite common to find, especially in persons with calculi, either during the sharp pains caused by the stone or after a surgical operation, papules, petechiæ, or plaques." During the same year Germain Sée reported a case of general eruption resembling that of scarlet fever and coming on after an operation. These two reports are often cited, especially by the French, as the first descriptions of surgical scarlet fever in the literature; but as neither author believed the rash in question to be really scarlatinal, the credit belongs to the English of being the first to attribute these post-operative eruptions to scarlatinal infection, and to trace a causative relation between the operation and the eruption. Sir James Paget, in 1864, in a clinical lecture on scarlet fever after operations, was the first to turn the attention of the medical world to this subject. He gave the details of a case which has since become classical:

A boy was operated on for stone in the bladder. Two days after the operation he had a general scarlatinal eruption, not more intense near the wound than elsewhere; there was no history of infection, no angina, and no desquamation. One month later he had a considerable hemorrhage from the kidney, followed two days after by a second eruption, accompanied by slight angina, and followed by desquamation.

Paget also stated that the previous year during an epidemic of scarlet fever he had observed 6 cases occurring after operations, which cases he considered not accidental, but due to a peculiar susceptibility to the scarlet fever poison consequent upon the operation. The disease, although true scarlet fever, was modified by the

operation, the usual incubation period shortened, the rash irregular, the angina or desquamation or both lacking. Following upon the publication of this lecture, a number of similar cases were reported; thus, Maunder, Graily Hewett, Callender, Martin, and Henry Lee claimed to have seen scarlatina follow various forms of trauma.

Thirteen years after his first lecture Paget collected and published his 10 cases of surgical scarlatina in his *Clinical Lectures*, and in an appendix to these lectures Howard Marsh added 8 cases, while Thomas Smith stated that he had seen 7 cases of scarlet fever following the operation of lithotomy in 43 children. Paget suggested two possible explanations of the part played by the wound; either it forms a port of entry for the scarlatinal poison introduced at the time of operation by direct inoculation from the hands of the surgeon, or else the poison is absorbed before the operation and remains latent until it is enabled to develop in consequence of the lowered resistance of the body caused by the operation. This latter hypothesis seemed to Paget the more probable. He thought that scarlatina following wounds was far from rare, and that the obscure cases of death occurring shortly after operation in children might be explained as due to such a latent infection.

Paget's views received strong confirmation in the eighteen months following the publication of his *Clinical Lectures*, through three reports of epidemics of surgical scarlatina in different hospitals. Goodhart and Paley collected from the records of Evelina Hospital 25 cases of scarlet fever, 18 of which were surgical cases. The infection was traceable in almost all of these; and all the cases as they developed were sent to the fever ward, where none of them subsequently contracted scarlet fever. These authors hold further that any inflammatory process, not necessarily wounds only, favors the development of the scarlatinal poison, which, entering in the usual way, finds the bodily resistance lowered and develops in consequence of the injury, but not directly through it. Stirling described an epidemic of scarlet fever in the Children's Hospital and one in St. George's Hospital, involving 33 cases in all. He believes that there is a real relation between trauma and scarlet fever, but rejects as unproved the explanation given by Paget; the theory that a lowered bodily resistance due to the operation allows the previously absorbed poison to develop is disproved by the fact that surgical scarlatina is as apt to follow an insignificant operation, such as the aspiration of an abscess, as a major operation.

The epidemic of surgical scarlatina described by Howse occurred in a surgical ward in Guy's Hospital and involved only 4 cases. Howse goes farther than the authors just quoted, saying that surgical scarlatina is much more common than is usually supposed, and that the rash usually described as septicæmic is really scarlatinal.

The French writers of this period, between 1864 and 1880, show themselves much more cautious than the English. Verneuil, at the

time when Paget's first lecture was creating so great a stir, wrote on various post-operative rashes caused by pyæmia, including among them the scarlatiniform rash. His pupil, Tremblay, also asserted that the post-operative rashes called by some scarlatinal are really pyæmic, even in those cases where the accompanying symptoms in many ways resemble those of scarlet fever. Terrillon, in 1877, expresses doubt as to the scarlatinal nature of "surgical eruptions." He quotes Braidwood's description of the rash of pyæmia as an "erythematous scarlet rash," appearing on the third to the fifth day after the initial chill, lasting from five to eight days and disappearing in the same order as it appeared. This description would apply to the rashes usually described as scarlatinal. In fact, Terrillon's case of septic erythema reads exactly like most of those reported under the name of surgical scarlatina.

Aulnas, in 1878, classes under the head of septicæmic eruptions those occurring after surgical operations, those occurring in the puerperium, and those occurring in the course of some internal suppurative disease, inasmuch as they are all due to the same pathological agent. Sanné, in Déchambre's *Dictionnaire*, 1879, states that scarlet fever may occur after trauma, but that such a case does not constitute surgical scarlatina. In his opinion, these cases are simple instances of infection anterior to the injury, and the relation between the wound and scarlet fever is merely one of coincidence. Two or three English writers also take the same stand; thus, Ffolliott, Braxton Hicks, and Dobbin all report cases of scarlatiniform rash following injuries, which they attribute to pyæmia. Cheadle, while admitting that patients with open wounds are extremely susceptible to scarlet fever, nevertheless maintains that a scarlatiniform eruption, not contagious, and not followed by the sequelæ of scarlet fever, appears in various traumatic conditions independently of the scarlatinal poison. Litten, in Germany, also describes a septic eruption so like that of scarlet fever that he knows no absolute means of differentiation. Desquamation may follow this eruption and may be quite like that of scarlet fever.

The study of surgical scarlatina received a new impetus in 1881 at the meeting of the International Congress in London. Riedinger, of Würzburg, reported 14 cases of surgical scarlatina which occurred when there was no medical scarlatina in the vicinity. He had no doubt as to the diagnosis in these cases, although the fact that severe inflammatory changes were present in 4 cases might have aroused his suspicions. The fact that the disease was apparently noncontagious in all but 1 case is also mentioned, but not as throwing doubt on the diagnosis. The clinical details are not very satisfactorily given; angina is mentioned in only 4 of the 9 cases; desquamation in 3.

Marsh, in a paper before the same Congress, supported Paget's theory as to surgical scarlatina, and pointed out the fact that the

disease had almost disappeared from the Children's Hospital since the building of an isolation ward for fever cases.

An important thesis written on surgical scarlatina in 1882 by Batut, a pupil of Trélat, has been overlooked by the majority of writers on this subject, and is not quoted even by those who hold similar views. Batut's contention is the same as Sanné's, that there is no relation of cause and effect between traumatism and scarlet fever, but only a simple coincidence. Like Stirling, Batut refuses to believe that a predisposition could be created by so slight an operation as the aspiration of an abscess. In his review of the cases published up to this time, he does not call in question the diagnosis, but only the assumed relation between the trauma and the development of scarlet fever. Batut states that he has followed in this thesis the views of his master, Trélat, who published 3 cases of surgical scarlatina in 1878 and 3 more in 1881, but who afterward came to regard the English theory as unfounded, and the relation between scarlet fever and trauma as accidental, not inherent.

Hoffa's critical review of the literature on this subject is frequently quoted. While accepting the English view he rejects most of the reported cases, admitting only those in which the eruption started at the wound. He makes, therefore, a sharp distinction between scarlet fever as an intercurrent disease and true surgical scarlatina, and he also warns against confusing the latter with the rashes due to vasomotor disturbance or to the absorption of poisons, especially carbolic acid.

Brown also insists upon a careful distinction between a scarlatinal rash proper and a scarlatiniform eruption resulting from sepsis or drug poisoning. He gives 2 instances of carbolic acid rash which are interesting, because in both he based his diagnosis entirely on the absence of sulphates in the urine, a point not mentioned by any other author, although many have described cases which had been freely treated with carbolic acid. (Henoch, Goodhart and Paley, Marsh.) Hoffa stated that desquamation does not follow the rash of carbolic-acid poisoning, but there was desquamation in Brown's cases.

In 1887 Murray published a description of an epidemic of scarlet fever in a surgical ward. There were 27 cases in the ward, 23 surgical and 4 medical. Six of the surgical cases took scarlet fever, as well as 1 of the 4 non-surgical cases. The proportion seems about the same, but Murray thinks that the increased susceptibility of the wounded is proved by the fact that of the 4 non-operated patients none had had scarlet fever previously and only 1 took it, while of the 23 operated patients, 5 had already had it and 1 of those 5 took it. The details given in these cases are very meagre, and the changes in the wounds are not noted; but only 1 case had been treated antiseptically. At the same time there were in the ward 6 other surgical cases which showed slight symptoms of septic infection, as anoma-

lous rashes, or a red area surrounding the wound; but these had all been dressed antiseptically and none had symptoms of any severity. Murray considers that the disease was the same in all—surgical scarlatina; but in the last series the action of the poison was entirely local, owing to the use of antisepsis.

A frequently quoted treatise on this subject is Koch's thesis in 1892. Koch reports 31 cases of surgical scarlatina, which occurred in the Kinderspital in Basel, 15 of them tracheotomy cases. He thinks a tracheotomy wound especially liable to scarlatinal infection, for it cannot be kept entirely aseptic, but is always more or less exposed to the air.

Two articles appeared in 1895 within a few months of each other, in both of which surgical scarlatina, while treated as a real affection, is made to seem only one manifestation of an infectious agent, which may in another patient cause erysipelas, puerperal fever, phlegmon, lymphangitis, or ordinary scarlet fever. Ricochon describes a series of cases which he claims were distinctly related, and in which the same infection took different forms in different persons; lymphangitis, scarlet fever, erysipelas, suppuration. Brunner quotes this series of Ricochon in support of his own theory that the same infectious agent can cause phlegmon, lymphangitis, lymphadenitis, and a scarlatinal eruption—that in fact these may be successive stages of the same process. Erysipelas also is due to the same infective agent. He quotes Lenhartz (*Jahrb. d. Kinderheilk.*, 1888), who states that Professor Heubner contracted facial erysipelas after examining the throat of a child with scarlatinal diphtheria; also Braxton Hicks, who states that scarlet fever may be contracted from women with puerperal fever.[1] Brunner thinks that changes in the wound similar to those seen in the tonsils in scarlet fever constitute proof that infection took place through the wound.

Two American physicians undertook to prove experimentally the possibility of infection with scarlet fever through a wound, although the principal object in both cases was to obtain a serum for therapeutic purposes. Ashmead used an adult Japanese, as the people of that race are apparently immune to scarlet fever. He inoculated this man with the scales from a young German who was in the twenty-fifth day of the disease. During the following two days the Japanese had a slight headache, and there was a mild inflammation of the forearm where the inoculation took place. On the third day a scarlet area appeared around the inoculation wound, with fever, redness of the fauces, and itching. The red area remained visible for several days, then faded, and on the eighth day began to desquamate. Stickler's more extensive experiments were made on ten white children, all of whom were in normal health at the time of

[1] In this connection may be mentioned Solly's statement quoted by Lea, that whenever he saw a surgical case becoming phlegmonous he expected to have scarlet fever or erysipelas break out in the same house.

inoculation; he used for inoculation mucus from the throat of a mild case of scarlet fever mixed with 1:600 of carbolic acid and made the injection into the arm. The results, which were published only after Stickler's death, are given in very condensed form and lack many details, and it is hard to know how much importance to attribute to his experiments. The reaction to the inoculation was singularly prompt and uniform, but the disease was not typical scarlet fever. Control experiments with the mucus of non-scarlatinal angina would be necessary to prove the specific nature of the reaction, for there is a strong probability that it was due to septic infection.

A careful and exhaustive review of this subject was given by de Bovis in the *Semaine médicale* for 1902. De Bovis has collected 150 cases from the literature, and although he is not ready to accept all of these, it is apparent that the scrutiny to which he subjects them is not oversevere. For instance, in cases where there is no description of the symptoms, he accepts the term "typical scarlatina" as sufficient. He thinks that the cases reported in the earlier literature, however meagrely, may be accepted also, because, in the pre-antiseptic days, surgeons must have been so familiar with the appearance of pyæmic eruptions that they could hardly have mistaken one for scarlatina. The great diversity of opinion, however, among the earlier writers does not bear out this view.

The most careful article which I have found in the English literature on this subject is Manning's "Skin Eruptions which Occur in Septicæmia following Scarlet Fever and Diphtheria." Without entering into the question of surgical scarlatina, Manning succeeds in indirectly throwing light on several vexed questions concerning it. His views are based upon a study of almost 6000 cases of scarlet fever and diphtheria treated in the City Hospital of Birmingham. In 2.1 per cent. of these cases a condition supervened which he explains as a toxæmia due to "tissue necrosis in septic surroundings" and identical with that often seen in cases of sloughing wounds, puerperal fever, or in the third week of typhoid fever. In his cases the cause was found in the ulcerative processes in the nose and throat. The attack usually came on during the second week, but might be as early as the fifth day or as late as the thirtieth. The first symptoms were: rise of temperature, restlessness, exacerbation of the throat symptoms, followed after about twenty-four hours by a scarlet or pink eruption, which, in the early stages, was in the form of spots with well-defined margins, disappearing on pressure. It appeared over the joints and prominent bony points, while a papular eruption appeared on the arms and legs. In mild cases the eruption faded and did not recur, but in severe cases it spread rapidly and by the second or third day resembled the rash of scarlet fever very closely. Albumin was often found in the urine. Profuse desquamation followed, accompanied by defervescence, and in the majority of cases succeeded by a second rise of temperature and a second

eruption spreading more rapidly than the first, and with an exacerbation of all symptoms. A second desquamation followed this eruption. Most of these cases were fatal, death occurring sometimes after the second rise of temperature, before the second eruption, sometimes after the second eruption, and due usually to septic pneumonia, diarrhœa, or exhaustion. In the non-fatal cases the eruption usually lasted only five days.

Rashes such as these, Manning says, are usually regarded as belonging to relapsing scarlet fever or scarlatinal infection complicating diphtheria, and when the patient is not under observation from the outstart, so that the early features of the rash can be noted, errors in the diagnosis are hard to avoid; for the fully developed eruption usually resembles that of scarlet fever, and the desquamation adds to the confusion. The important point to notice is the distribution of the rash in its early stages. It would be interesting to know how Manning, in the light of his experience, would explain such cases as the classical one of Paget's with its two eruptions, or Brown's case of circumcision followed by gangrene, or Beaudouin's similar case. Verneuil's case of septic eruption had the eruption first over the joint surface.

The literature on the subject of surgical scarlatina thus briefly reviewed comprises 174 cases, the details of which are given more or less fully. To these may be added 10 cases from the records of the Hospital of the Memorial Institute for Infectious Diseases, in Chicago, between October, 1902, and March, 1903. These cases appeared in the Presbyterian Hospital and were transferred to the Memorial Institute.

The first 2 cases, one an operation for tuberculous epididymitis, the second an operation for tuberculous inguinal glands, developed within six days of each other; and a non-surgical case, a male attendant, also developed scarlet fever during this time. The latter had a typical attack with characteristic rash, angina, palpable cervical, epitrochlear and inguinal glands, and characteristic desquamation. The same was true of the second surgical case. The first surgical case, however, was not typical, and, except for the fact that the attendant was apparently infected from this man, his disease would probably not have been diagnosed as scarlet fever, for a sore throat and slight fever were the only symptoms noted until the illness of his male attendant six days later led to a careful examination, when a faint rash was found in the inguinal region, and he was transferred to the Hospital of the Memorial Institute. Later on there was slight desquamation. The wound suppurated for more than three weeks.

No other cases developed in the Presbyterian Hospital until December 22d, when a boy, aged thirteen years, under treatment for tuberculous hip-joint, without abscess, took scarlet fever in the children's ward after a sojourn of three weeks in the hospital. Rash,

angina, strawberry tongue, swollen glands, albuminuria, and desquamation were all present in this case.

Between March 1st and March 6th, 7 children developed scarlet fever in the children's ward. Six of these were surgical cases between the ages of three and ten years, and the operations were as follows: cleft palate, 2; osteotomy, 1; removal of tuberculous glands from the neck, 1. The other 2 were in plaster-of-Paris dressings for tuberculosis of the spine, and for congenital dislocation of the hip-joint. In all 6 cases there was a rash, swelling of the cervical, inguinal and epitrochlear glands, and desquamation. Angina was absent in both of the cleft-palate cases; albuminuria was absent in all. The osteotomy case was the only one in which there was any interference with union in the wound. Slight suppuration along the line of incision persisted in this case throughout his stay in the hospital.

In December, 1903, a child died of scarlet fever with septic complications. The interne, who had been in constant attendance on the child, assisted at the autopsy, and on the following day noticed pain and redness in the middle finger of the right hand. The next day there was suppuration in the finger, sore throat, general malaise, and a temperature of 102° F. Yellowish patches appeared on the tonsils, which were much swollen. On the third day a faint rash was observed on the upper part of the chest, the neck, and above the scapulæ, and the temperature was 103° F. The finger continued to suppurate for four or five days, and slight scaling appeared on the neck. One week later a scarlet rash appeared on the lower part of the abdomen, which, however, was not followed by scaling. A mild nephritis developed in the course of the third week. Streptococci were isolated from the blood and organs of the boy and from the throat and inflamed finger of the interne. The prosector who performed the autopsy developed a streptococcal sore throat on the day following the autopsy.

These 10 cases might be regarded as surgical scarlatina—certainly they are as typical as many in the literature—or, the first 9 might be diagnosed as scarlet fever occurring in surgical cases, the last as streptococcal infection through an abrasion of the finger. As we have seen, there is ample authority for either of these views.

There are two chief points to be considered in deciding this question: Is the disease reported in the literature as surgical scarlatina really scarlatina? If it is scarlatina, what is the relation between the injury and the disease?

Of course, if we accept the opinion of most earlier writers, and of Fürbringer, Gomprecht, Ricochon, and Brunner among the later, we shall believe scarlatina, whether medical or surgical, to be only one manifestation of the same infection which causes erysipelas and various septic processes. But until it is proved that the streptococcus is the cause and not a complication of scarlet fever, we must continue to believe that the infective agent concerned in scarlet fever,

a disease followed by immunity, is not the same as the infective agent concerned in the large number of septic processes which are never followed by immunity. We are not ready to believe that erysipelas, septicæmia, and scarlet fever are the same disease, however similar their symptoms may be. That the symptoms are similar is undeniable. A cursory comparison of the two classes of cases in the literature, those of "surgical scarlatina" and those of "septic rash," is enough to convince one that the same affection has been given different interpretations by different observers. Scarlet fever, if it is a single disease—Gerhardt hazards the bold hypothesis that it is really a group of diseases—is admittedly irregular and polymorphous in its manifestations. It is hard to find two authorities who agree entirely on the cardinal symptoms which must be present to make up a real case of scarlet fever. The rapid onset of the symptoms; the early appearance of a characteristic rash, first on the chest and neck, then rapidly becoming general; the angina; the swelling of the lymphatic glands; the nephritis, and the desquamation make up the typical picture of scarlet fever.

But we may have scarlatina sine febre, sine exanthemate, or sine angina. Jürgensen cites 2 cases of the first; Leichtenstern, 1. The absence of angina in a case with a well-marked rash is rare, but does occur, and the absence of exanthem has also been observed. As for the swelling of the glands, we have Schamberg's statement that the inguinal glands are swollen in 100 per cent., the axillary in 96 per cent., and the maxillary in 95 per cent.; but, judging by the ordinary literature, one would conclude that swelling of any glands, except the submaxillary and post-cervical, was a great rarity, for it is almost never mentioned. Nephritis is insisted on by some as a cardinal symptom, but by many others regarded as a complication only, an instance of infection by pus organisms, and no more essential to the disease than the suppuration of the cervical glands.

Finally, desquamation, which is a tardy symptom at best, may be scanty or atypical, and, even if it is typical in character, proves very little, because it follows other eruptive diseases also, as erysipelas and erythema scarlatiniforme. Jürgensen would lay greater stress on the immunity conferred by a single attack of scarlet fever than on any other symptom, and would, therefore, reject, unless the evidence were overwhelming, all cases in which there was a history of previous scarlet fever or in which there were relapses. This persistent immunity after a single attack, together with the transmission of the infection to others, he regards as the most positive points of evidence.

Given this variety in the symptomatology of scarlet fever, what are the diseases which, in a wounded person, are most apt to resemble it and be confused with it? I say in a wounded person, because the fact that a wound is present complicates the diagnosis very seriously. Jürgensen speaks with great emphasis of the difficulty of differen-

tiating between scarlet fever and sepsis, even in patients without injury or operation; how much more difficult does the question become when an injury is present, and when the symptoms evidently depend in some way upon it?

The surgeons and the dermatologists have much to say on the subject of eruptions which occur in the course of toxic and septic processes, although, except for the French writers, it is seldom that one finds any one of them suggesting that this might be a way of accounting for some of the cases of surgical scarlatina.

Erysipelas is probably the least likely of all the post-traumatic eruptions to be mistaken for scarlet fever, yet Holmes, early in the history of "surgical scarlatina," asserted that most of the eruptions so described were due either to erysipelas or pyæmia. Triwousse was unable to decide on the character of the eruption in his patient before the transmission of the infection in typical form to another person showed that it was erysipelas; his second case, also diagnosed as erysipelas, reads very much like the surgical scarlatina cases of Hoffa, Stirling, Howse, Gimmel, and Ingerslev.

The general symptoms of erysipelas are those of septic poisoning and may simulate closely those of scarlet fever, for the infection often spreads to the mucous membrane of the pharynx, causing either a dry, shining, red appearance, or œdema with swollen tonsils and membranous patches. One cannot help noticing in this connection how often the red throat without the typical scarlatinal changes is mentioned in the cases of "surgical scarlatina" in the literature. The wound may suppurate and become gangrenous; or, as is not uncommon, it may heal rapidly. Bollici's cases are somewhat suggestive of erysipelas. In his first and second cases there were bullæ and blebs around the wounds, in which there was no suppuration; the eruption began in red patches and there was great itching. In his third case the eruption was vesicular, with large blebs around the wound and over the abdomen. Riedinger diagnosed one of his cases as erysipelas, apparently because of the character of the eruption; but when albuminuria, swollen tonsils and desquamation followed, he decided that it must be scarlet fever. The fact that desquamation often follows erysipelas and that the scales are highly infectious has undoubtedly helped in confusing these two diseases.

The rash occasionally seen in diphtheria, especially in cases of mixed infection with pus organisms, is not very uncommon. The authorities are not agreed as to its frequency; Germain Sée found it in 22 per cent.; Mussy says 12.5 per cent.; Cadet de Gassicourt and Sanné only 3.5 per cent. If the views of these authors were compared as to the frequency of "scarlatina complicating diphtheria," the difference of opinion among them might become clearer. At all events, it is an established fact that an erythematous eruption, accompanied by a rise of temperature, may occur at any stage in

diphtheria, that it is frequently very like the rash of scarlet fever, if not indistinguishable from it, and that it is most apt to occur in cases which show symptoms of septic poisoning (Manning). If we examine closely the often-quoted tracheotomy cases in Koch's thesis, the conviction is forced upon us that the symptoms in many of them point either to septicæmia with diphtheritic inflammation and suppuration in the wound, or to multiple embolic infection with slight changes in the wound, but with symptoms of suppuration in the internal organs. In these cases the tracheotomy wound probably permitted the pus organisms, formerly localized in the throat, to escape into the general circulation.

Even more difficult is the diagnosis between scarlet fever and the rashes which appear in septic and pyæmic conditions or as a result of poisoning by drugs. It is to the French that we owe the most thorough work on scarlatiniform and scarlatinoid eruptions. As long ago as 1876 Féréol reported to the Hospital Society of Paris a case of "erythéme desquamatif scarlatiniforme récidivant," in which the eruption closely resembled that of scarlet fever. The tendency to relapse in Féréol's case was so great that it seems to have overshadowed the other symptoms; and this may be the reason that similar cases which had not as yet any history of relapse were not reported as belonging to the same class. At all events, there were very few instances of this disease recorded for years after Féréol's observations. One is mentioned in Allbutt's *System*, vol. viii. p. 541, in which the present attack was the only one, and no relapse had as yet occurred. The author believes that such cases are usually diagnosed as scarlet fever, which was also true of the one which came under his observation. It was an article by Besnier, in 1890, which first made this particular form of erythema known to the general medical world. Besnier and most of the French writers after him distinguish two forms—namely, the "scarlatinoid" and the "desquamatif scarlatiniforme." The "scarlatinoid" simulates scarlet fever very closely, for it comes on rapidly and has more fever, involvement of the viscera and mucous membranes, and more serious results than the "scarlatiniforme," which is apt to be subacute and to last longer; Féréol's case belongs to this latter class. The English and American dermatologists consider this distinction on the ground of acuteness, pyrexia, and duration quite unnecessary and disadvantageous, and they therefore recognize only one disease, erythema scarlatiniforme.

According to the text-books, this disease is "a non-contagious eruption, which simulates true scarlet fever, but differs from it in having most diverse causes instead of one cause"; "a febrile affection occurring idiopathically or in association with septicæmia, pyæmia, autotoxæmia, or drug or food poisoning, its most distinguishing features being the scarlatina-like eruption, the non-contagiousness, and the tendency to recurrence." The fever is apt to

be less high than in scarlatina, the tongue is furred or dry, red and glazed, the angina is mild, and the eruption is apt to appear early and develop rapidly. Desquamation also appears early, sometimes before the eruption has quite faded, and in some cases a second eruption comes on before the desquamation of the first is complete. (Compare Schäffer's case of "Tetanus with Relapsing Scarlatina," also Manning's statements as to septic rashes in scarlet fever and diphtheria.) It is usually more intense than the desquamation of scarlet fever and more apt to cause the shedding of "gloves" and "socks" from hands and feet, and to involve even the nails and hair. (Compare the case of Riedinger, intense desquamation involving two nails; Stickler's case, the hands desquamating twice.) Relapses are the rule, but there are cases reported in which there was only one attack.

The causes of erythema scarlatiniforme are:

1. Reflex vasomotor disturbances after operations on parts rich in nerve supply. This reminds one of the testimony of the early writers and of Hoffa as to the frequency of eruptions after operations on the genito-urinary tract.

2. Autotoxæmia from absorption of the products of tissue destruction after crushing or lacerating wounds. (Compare the third case of Brown, a lacerated wound of the leg; Braxton Hicks' case, strain to the muscles of the hip; and Cameron's third case, crushing of the hand.)

3. Poisoning from drugs, the most important for our purpose being carbolic acid and mercury, so freely used in the early days of antiseptic surgery. Brown's cases of carbolic acid poisoning and Hoffa's case of mercurial poisoning might by other observers have been considered cases of scarlatinal rash.

4. Septicæmia, pyæmia, general gonorrhœal infection, puerperal septicæmia, diphtheria. The diagnosis is not difficult in the absence of a wound, injury, or suppurative process, for then the slight constitutional symptoms, slight involvement of the throat and absence of "strawberry tongue" would serve to distinguish it from a well-marked case of scarlet fever. But if there is already an injury, especially one with septic infection, the question becomes quite different.

The general symptoms of septicæmia are too well known to need recapitulation; but mention may be made of the frequent occurrence of lymphangitis leading from the wound, swelling and tenderness of the lymph glands, the fact that serious general symptoms may follow insignificant wounds, and that the changes in the wound may be profound or very slight.

Brown's case, often quoted as a striking instance of surgical scarlatina, suggests the picture of rapid septic infection following a slight operation. A medical student was operated on for phimosis. At the time of the operation he was hot and perspiring, but presented

no other unusual symptoms. Thirty hours after the operation his temperature was 104° F., he was comatose, and the body was covered with an abundant scarlet rash. There was no angina. The wound became rapidly gangrenous, and death occurred seventy hours after the operation. Inquiry revealed the fact that he had been paying daily visits to a scarlet fever patient for two weeks before his operation.

Bollici thought the diagnosis of pyæmia in his and Moreschi's cases ruled out because there was no pus in the wounds; but the literature of surgery is full of instances in which, with small wounds secreting perhaps only a little yellowish serum, a general pyæmia occurred from lodgement of emboli in the joints, bones, lungs, kidneys, etc. Such a pyæmia may follow the opening, spontaneous or operative, of an old abscess or empyema, or even a simple aspiration, the pus organisms, which were formerly walled in, now being able to escape into the general circulation through the ruptured tissues. There is a very large number of old abscesses among the cases of "surgical scarlatina" in the literature. Long-continued suppuration preceding or following the operation or both are mentioned in 29 cases; gangrene, diphtheritic inflammation, œdema, blebs, and fetid odor in 20 cases.

In such cases the diagnosis between sepsis and scarlet fever becomes very difficult, and must rest largely upon (*a*) the non-contagiousness, as shown by the absence of any history of exposure to scarlet fever or of transmission of the infection to others, together with (*b*) the tendency to relapse, (*c*) the absence of a typical strawberry tongue, and (*d*) an unusually early and long-continued desquamation.

(*a*) As to the non-contagiousness, if we omit Stickler's 10 experimental cases and the 97 concerned in the hospital epidemics, we shall find that only 15 of the 67 remaining have a clear history of exposure to scarlatinal infection, and only 8 have a history of transmission of the infection, although in many cases there was ample opportunity for such transmission; and among these 8 cases some are open to question, for the history of the infected patient is sometimes strongly suggestive of septic rather than scarlatinal infection. (See May's cases, Brown's third case, Hoffa's second case, Riedinger's fourth case.) In others the apparent transmission may have been only a coincidence, as an epidemic existed at the time. (Lea's second case, Beaudouin's case.)

It is hardly probable that the authors would have omitted to mention such important contributory evidence as a history of exposure to infection, or of transmission by infection, if such evidence had been at hand. In several instances an apparent non-contagiousness is noted; "the patient, although not isolated, did not infect any of the other children; . . . there was at the time no scarlet fever in the vicinity." (Riedinger, Strubell, Perlis, Lea, etc.)

(*b*) Relapses or recurrences are noted in Schaeffer's case, in Konetchke's, and in Paget's. Page's case had had scarlet fever twice before, the last attack being only seven months before this one. Brunner's case, one of Murray's, and one of May's had had previous attacks.

In considering the question of the tendency to recurrence in erythema scarlatiniforme, it must be remembered that there is necessarily always a first attack, and that many of these cases were reported soon after their occurrence, before it was possible to know whether or not there would be repeated attacks; others were hospital cases and passed out of the sight of the physician who observed the first attack. Even if there was but one eruption without any recurrence, we might still insist that the case might possibly be non-scarlatinal; for there are on record instances of erythema in which there was but one attack, and which, by their non-contagious character and excessive desquamation, were shown to belong to this class.

(*c*) A "strawberry tongue" is mentioned in only 17 of the 174 cases; more often, especially in those which are most strongly suggestive of septic infection, the tongue is described as furred or coated.

(*d*) The facts as to the time of appearance and length of duration of the desquamation are harder to obtain. In only 46 cases is the length of time given between the appearance of the eruption and the beginning of desquamation; the duration of the desquamation is given in only 16 cases. In 19 cases desquamation began on the seventh day or earlier; in 11 it was on the fifth day or earlier. Brunner's case, Ingerslev's second, and Koch's fourth and seventh cases began to desquamate as early as the fourth day; only 14, on the other hand, began later than the eighth day. It is not possible to find any positive data as to the time which elapses between the eruption and the desquamation in typical medical scarlatina. De Bovis gives the average as the eighth day or slightly later for medical scarlatina, the eighth day or earlier for surgical, and our figures would bear out this statement. Desquamation lasted seven days or more in all the 16 cases in which the duration is given; in 2 the time was twelve days, in 1 thirteen, in 1 fourteen, in 3 fifteen, in 1 nineteen, in 1 twenty-one, and in 1 forty days. (D'Espine and Marignac.) Lannelongue's patient is an instance of a rapidly developing rash with long-continued desquamation. In Schaeffer's case the second eruption lasted eight days, the desquamation twenty-one.

De Bovis lays stress on the slight precocity of the desquamation in surgical scarlatina, and says that in some cases there is no desquamation at all. According to him, this is one of the five points of difference between surgical and medical scarlet fever; the other four being the large proportion of adults affected, the slight angina or absence of angina, the short period of incubation, and the fact that the

eruption is apt to start in a peripheral part near a wound, and to spread in the manner of a lymphangitis.

As it happens, all these points of divergence from true medical scarlatina are points in the diagnosis of erythema scarlatiniforme. This latter eruption, when caused by toxæmia or septicæmia, may come on a few hours after an operation of any kind, or at any time in a long-continued suppurative disease; hence the unusually short or unusually long "period of incubation," namely, the length of time between the injury and the eruption. Erythema scarlatiniforme does not cause a serious angina; redness and swelling only are the usual changes; hence the "slight angina" of surgical scarlatina.

The eruption may begin in any region, and it will be remarked on looking over the cases in the literature that the eruption was very often atypical in its place of origin, even in those cases where it bore no direct relation to the wound. Frequently, also, it was not general, but confined to certain localities, as is true of erythema scarlatiniforme.

The occurrence of lymphangitis is almost indisputably a point in favor of the diagnosis of septic rash. The large proportion of adults attacked is explained by the fact that injuries and operations of all kinds are more common in later life than in childhood, and, therefore, septic rashes are also more common. Finally, de Bovis' statement that the desquamation of surgical scarlatina is more precocious than that of medical scarlatina, and in many instances lasts longer, is, as we have seen, a strong bit of evidence in favor of erythema scarlatiniforme.

It seems, therefore, justifiable to rule out as unproved all those cases in the literature of surgical scarlet fever in which the presence of toxæmia or septicæmia was highly probable and in which the symptoms deviated more or less from those of typical scarlet fever, but not from those of toxæmia or septicæmia.

There remain, however, after the elimination of these doubtful cases, a large number which seem to have been indubitably cases of true scarlet fever. Are these cases of "surgical scarlet fever," or of "scarlet fever in the wounded?" In other words, is the relation between the wound and the disease one of causation or of coincidence?

When we examine the literature for proof of Paget's widely accepted theory that injuries of all kinds predispose to scarlatinal infection, we find that they are distinctly unsatisfactory. There is a general impression, especially among surgeons, that scarlet fever is more apt to occur in wounded persons than in the unwounded, but it is impossible to find convincing proof of this opinion. It cannot be found in the reports of hospital epidemics, not even when medical and surgical cases were kept in the same wards. Goodhart and Paley give no comparative statistics. Murray's cases are so

imperfectly reported and so strongly suggestive of septicæmia that they may be ignored. Howse's epidemic was in the surgical wards. Stirling tried to establish from hospital statistics evidence of an increased susceptibility on the part of operated cases as against the non-operated, but was unable to do so.

Koch's cases seem at first sight convincing, for the number of tracheotomized children who developed scarlet fever symptoms was much in excess of the non-tracheotomized. But if we exclude those which showed symptoms resembling sepsis, such as suppurative and diphtheritic inflammations of the wound, with foci of suppuration in lungs, kidneys, etc., we shall have reduced the number appreciably; and it may be still further reduced, in view of the fact that an erythematous eruption may occur in the course of diphtheria, even where there are no severe septic complications. In his second series of cases Koch gives no statement as to the number of operated and non-operated cases in the wards.

Much is made of the fact that the epidemics of scarlet fever among the surgical cases in hospital wards gradually ceased after a more complete system of isolation was practised. This must also have been true of the medical cases of scarlatina, and if there has been a gradual diminution in the cases of post-operative rash, it may be explained by the fact that the advance in antiseptic methods and in isolation of septic cases has gone hand-in-hand with the advance in isolation of fever cases. From time to time the question is raised why scarlet fever should be the only infectious disease to enter through a wound, and more rarely the assertion is made that this is not true, that other infectious diseases act in this respect as scarlet fever does, but are overlooked for various reasons. The references to measles in wounded persons are very infrequent, but there are two possible explanations for that. Either, as Goodhart thought, surgical measles is less striking than surgical scarlatina, and simply has not attracted so much attention; or else, which seems more probable, measles is not so apt to be confused with other eruptive diseases as is scarlet fever, and, therefore, the literature of "surgical measles" has not been increased by the addition of cases of septic rash as has certainly been true of the literature of "surgical scarlatina."

Many writers, especially Brunner and de Bovis, consider that there is an additional proof of the scarlatinal infection having entered by the wound when the latter shows inflammatory changes, especially when there is necrosis, diphtheritic inflammation, or gangrene. But this is a proof which can be made to work the other way; for, if there is such a thing as septic scarlatiniform rash, a fact admitted by all, then these would be the very cases in which one would look for such a rash.

There are certain cases which are often quoted as proof that a wound may render susceptible to scarlet fever a person who has

hitherto been apparently immune. Such are v. Leube's[1] famous case and the 3 similar cases of Riedinger, all of them physicians, who had been frequently exposed to scarlet fever infection during childhood and in the course of their practice, but were apparently immune until after they had received an injury. In 3 cases the injury was an autopsy wound; in the fourth a pin-prick from a surgical dressing. The 3 cases reported by Riedinger could not have become infected with scarlet fever at the time of the injury, for the autopsies were not made on scarlet fever patients, nor was the bandaged man a scarlet fever patient; indeed, Riedinger particularly insists on the fact that at that time there was no scarlet fever anywhere in the vicinity. We have already seen that Riedinger's cases are among the most unsatisfactory in the literature from the point of view of surgical scarlet fever, and it is unnecessary to discuss them further in this connection. This is not true, however, of v. Leube's case, which is unanimously accepted as the most if not the only indisputable case in the literature. It would require much boldness to refuse to accept this case, although the same clinical picture might be presented by a case of septic poisoning from an ordinary autopsy wound. It may also be suggested that, supposing the disease to have been surgical scarlatina, the fact that v. Leube's apparent immunity was overcome by a slight injury was not absolutely proved; for instances are on record of unwounded adults succumbing finally to an infectious disease, even when they had been repeatedly exposed before without contracting it. Jürgensen tells of a sick nurse of whom this is true. Unless, therefore, this case of v. Leube's is to be regarded as sufficient in itself to prove the causative relation between wounds and scarlet fever, it must be admitted that the literature on this subject does not as yet present such proof.

Conclusions. There are about 174 cases of surgical scarlatina reported in the literature more or less in detail, and passing mention is made of many more. Operations and wounds of all kinds, as well as inflammatory processes, are supposed to predispose to scarlatinal infection.

Surgical scarlatina is said to differ from medical scarlatina in five ways: 1. The large number of adults attacked. 2. The shorter period of incubation. 3. The mild angina or absence of angina. 4. The fact that the eruption begins at the wound or in some other unusual region. 5. The slightly precocious desquamation.

Proof of the existence of a special form of scarlatina occurring in connection with wounds rests upon the large number of cases recorded in which an eruption resembling that of scarlet fever fol-

[1] V. Leube had been frequently exposed to scarlet fever in childhood and in his practice. During an autopsy on a severe case of scarlet fever he wounded his finger, and the wound became painful. On the seventh day he grew ill with fever, vomiting, and angina. On the eleventh day a scarlet exanthem appeared, first in the form of a broad, red stripe from the finger up the arm, then spreading over the body. Desquamation began on this arm.

lowed some injury, and upon the frequency of epidemics of scarlet fever among surgical cases, in the days before complete isolation of fever cases was practised.

Careful examination of the reported cases confirms the view held by most French writers, that the eruption in question is often due to septic infection and is not scarlatinal; and that the variations of so-called surgical scarlatina from the typical form of scarlet fever are not due to the unusual mode of infection, but to the difference in the infectious agent.

In the cases where the disease was undoubtedly scarlatina, there is no convincing evidence that the relation between the wound and the scarlet fever was anything more than one of coincidence.

The statistics of epidemics of scarlet fever in surgical wards in hospitals lack the necessary comparative statistics as to the same disease in the medical wards.

There is, as yet, no convincing proof in the literature that surgical scarlatina is anything more than scarlet fever in the wounded.

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THE INFLUENCE OF DAYLIGHT IN THE PROGRESS OF MALARIA,

MORE ESPECIALLY IN CONNECTION WITH QUININE TREATMENT.

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Dr. A. F. A. King nearly twenty years after he had hypothetically put forward and supported the theory of transmission of malaria by mosquitoes[1] has brought out a new and very interesting hypothesis concerning the biology of plasmodium malariæ,[2] an hypothesis which, as far as I know, has not hitherto been made a subject of discussion in any medical journal or publication, although in it King tries to inaugurate a revolution in the generally accepted treatment of malaria.

I first came across Dr. King's papers when I recently went through the medical publications in order to find, if possible, any casual com-

munication speaking for or against an hypothesis the possible correctness of which would indicate a treatment of malaria entirely opposite to the one proposed by King. King's arguments have, therefore, particularly interested me, but, as they have not convinced me, the following remarks which I should like to bring forward in connection with this matter take the form of an opposing criticism.

Dr. King thinks that he can explain a number of the clinical and epidemiological peculiarities from the supposition that the sporulation of the *plasmodium malariæ* cannot take place in the dark, but only in light, more especially red light, and he, therefore, proposes to treat malarial patients in the dark or in rooms with violet or purple window hangings; in other words, by a negative phototherapy similar to the Finsen treatment of smallpox.

Before I enter upon a more theoretical argumentation, I will refer to some experimental investigations by Harrington and Leaming[3] upon the effect of light on *amœba proteus*, which King takes as being in favor of the correctness of his hypothesis. The two doctors just referred to sum up that part of their experiments, which is of interest in this connection, in the following points:

1. Amœba streams in the presence of red light.

2. Streaming is retarded, stopped, or reversed by rays from the violet end of the spectrum.

3. Further, the effectiveness of the following kinds of light as inhibitors of protoplasmic flow diminishes in the order named: white, violet, red.

From these experiments King draws the conclusion that red light promotes the vital processes of the amœba, while violet light retards them; and, as *plasmodium malariæ* is a naked amœba, he infers that the same thing applies to the latter. Harrington and Leaming, however, distinctly point out in their essay that the streaming of protoplasm in the amœba is again continued after remaining a few minutes in any quality of light, and the point seems, consequently, to be the sudden change of light. Unfortunately, nothing is said about the intensity of the light employed, but, as far as one can judge, it has been comparatively faint, and the experiments seem mostly to point to the fact that a transitory rigor is produced by the sudden change from a light with less refrangible rays to a light with more refrangible rays, or from less intense light to more intense. Only one isolated experiment speaks in favor of a direct inciting effect of the red rays: a sudden change from darkness to red light brought about the movements referred to in the course of ten seconds; in this experiment it is, however, not excluded that the heating effect of the rays has been of importance. The assumption of this inciting effect of red rays (in contrast to those with shorter waves) is in any case not confirmed by the somewhat similar experiments which Dreyer[4] has carried out at the Finsen laboratory. In these

the blue-violet rays were found, in fact, to have an inciting—and in a powerful concentration a destroying—effect, while the amœbæ in the red light remained in about the same condition as in the dark. According to Verworn[5] and Davenport,[6] *amœba proteus* is not at all photopathic. The latter, however, found that the amœba showed itself negatively phototactic to light of an intensity varying from strong diffuse daylight to direct sunlight.

In accordance with the above, I cannot, in Harrington and Leaming's experiments, find any support for King's supposition that the *plasmodium malariæ* requires red light in order that its sporulation may be brought about.

1. King first opposes the supposition which has hitherto been generally accepted, that the warmth of the sun, as such, influences the frequency of cases of malaria, independent of the fact that a certain degree of warmth is, of course, necessary in order that the infection-carrying mosquitoes can live and preserve their activity; in this connection he refers to statements by various writers, and mentions, among other things, that negroes, whose dark skin in a particularly high degree absorbs the heat rays of sunlight, possess relative immunity from malaria. When, all the same, there exists a correlation between intermittent fever and warm climates or seasons, we must, according to King, look for the cause not in the warmth of the sun, but in the light of the sun, upon which depends the sporulation of the plasmodiæ.

All the conditions upon which the supposition was previously based, that warmth is an important factor in calling forth intermittent fever, may, with the same right, be held to be in favor of the view that sunlight plays a part as an etiological factor. When King, however, points out that the inhabitants of the island of Tahiti, according to Quatrefages, are free from swamp fever, although the island is only 18 degrees removed from the equator, and holds that this fact speaks against the theory of warmth being of importance, the same example may just as well be taken as a proof that neither can sunlight be the decisive factor. A more likely supposition, it seems to me, is that the conditions and nature of the soil are unfavorable for the development of anopheles, in the same way as they, in Denmark, for instance, have gradually become so by the reclaiming of swampy land by drainage, etc.

2. Febrile paroxysms appear, as a rule, in the daytime, and only in exceptional cases during the night. Even if febrile attacks at night occur somewhat more frequently than King supposes, it can hardly be denied that they occur with predominant frequency in the daytime, and this circumstance really does seem to speak in favor of daylight in some way or other being able to influence *plasmodium malariæ;* but as long as we have no accurate knowledge of the duration of the sporulation process of the plasmodiæ, the above fact does not tell us anything definite as to whether this effect of light is favorable or the opposite.

3. The fact that negroes are less frequently attacked by malaria than human beings of the white race, King attributes to the dark color of their skin, which hinders the light from exercising its sporulation-inciting influence upon the plasmodiæ in the blood. Even if it is not out of the question that the relative immunity of the negroes may be due to entirely different causes, it must be admitted that King has a good argument in favor of the correctness of his hypothesis; there is, however, every reason to accentuate, as Chappel[7] does, that the clothing of civilized nations as a protection against daylight may, in many cases, to some extent, be taken as an equivalent to the dark but often uncovered skin of the negroes.

4. King next quotes a number of writers, according to whom the frequency of cases of illness in malarial districts decreases during periods of much rain or mist, and he connects this decrease with the lesser intensity of daylight at such periods. I must confess it seems to me more natural to explain this by the supposition that rain and mist diminish the chances of being infected by the stings of anopheles.

5. King, after having drawn attention to various examples of spontaneous cure of malaria, and suggested that the patients in question had perhaps been treated in especially dark hospital rooms, puts forward the following hypothesis: The red blood corpuscles in which the plasmodiæ live gradually increase in size so that some of them are at length retained in the capillaries of the skin, where sporulation is then brought about under the influence of daylight. The fever paroxysm which accompanies this process is followed by the sweating stage, during which the bloodvessels of the skin are dilated, whereby the blood corpuscles therein retained are released and the new generation of plasmodiæ is distributed to the other organs of the body.

King looks for the potent principle of those medicines which have been successfully used against malaria in their ability to alter the optical peculiarities of the blood; some of them (Prussian blue, methylene blue) so color the blood that the rays of red light are kept away from the plasmodiæ, while others (quinine, esculin, fraxin) have the peculiarity in common of being fluorescent, and thereby *accentuating* the violet rays of the spectrum in the blood! According to some investigations, now more than thirty years old, by Jones,[8] Rhoads and Pepper,[9] the blood and most of the tissues in human beings and animals are slightly fluorescent; in malarial patients this fluorescence is considerably reduced, while it again increases by the taking of quinine. King sees in this proof that the healing effect of quinine upon malaria is due to its fluorescence; he seems, however, to attach more importance to the beneficial effect of red rays upon the plasmodiæ than to the detrimental effect of violet rays, and he recommends the following treatment of malaria: "Keep malarial patients in dark rooms or in rooms with

violet or purple windows; clothe them with garments impenetrable to light. Give the patients such remedies as render the blood dark or violet or lessen its translucency."

After this condensed summary of King's theories and argumentation, I will now proceed to mention some experimental investigations by other writers and some views which, as they will indicate an entirely opposite therapy, may possibly be of interest in this connection.

As is well known, Tappeiner[10] and his pupils have, in the course of the last five years, undertaken a series of most important investigations upon the poisonous effect of various fluorescent fluids in light and in darkness, respectively. It transpires from these investigations that the destroying effect of the substances in question, for instance, upon infusoriæ, is enormously increased by the effect of light. Even if the fluorescent fluids are so much diluted that in the dark they have no apparent detrimental effect upon infusoriæ, the latter, all the same, will be speedily destroyed in such solutions if they are placed in the sunlight or simply in diffuse daylight, which under normal conditions would not lower their vitality. It has been shown that the most effective rays in this respect are those which produce the strongest fluorescence in the fluids in question. I shall not here enter upon the exceedingly interesting question of rendering micro-organisms and animal tissue sensitized to light;* but only point out that the discovery of these phenomena happened during some experiments with the toxicity of various preparations derived from quinine, and that the quinine preparations themselves possess such sensitiveness-arousing qualities.

The surprisingly strong microbicidal effect of which rays otherwise fairly inactive at these and later experiments have proved themselves possessed, with regard to "sensitized" micro-organisms, has led me to look for the cause of the special effect of quinine preparations upon malarial patients in *the power of these preparations to make the plasmodiæ sensitive, so that the latter are destroyed or weakened under the effect of daylight.*

I will now briefly point to some of the circumstances upon which I think I can base this supposition:

1. The special effect of quinine in malaria can hardly be explained as a direct outcome of its toxicity with regard to plasmodiæ, although this undoubtedly is very considerable, and if we look for an explanation of this effect we must not leave out of consideration a peculiarity so distinct as the power to make micro-organisms sensitive in relation to light.

2. Quinine preparations have decided sensitiveness-arousing qualities. Ullmann's[11] investigations show, for instance, that paramæciæ, which are placed in solutions of quinine of 1: 20,000, first die after

* I have dealt comprehensively with this subject in Mitteilungen aus Finsen's medicinske Lysinstitut, 1904, vol. viii. Verlag, Gustav Fischer, Jena.

about five hours when standing in the dark, while they are killed in the course of eight minutes if placed in sunlight, under conditions which generally have no baneful effect upon paramæciæ.

3. According to Jacobson's[12] and Dreyer's[13] experiments, light, even after having passed through a layer of animal tissue, can exercise its microbicidal effect upon the sensitive-made organs. The depth at which it may be possible to obtain an effect will, of course, among other things, depend upon the intensity of the light.

4. The tissues of the human body are pellucid, and even if only a comparatively small portion of the surface of the body is exposed to light, the blood, and with it the plasmodiæ, will, on account of its continuous circulation, all the same be affected by the light.

If the correctness of the views I have here put forward should be confirmed by clinical experiments, *they indicate the advisability of treating malarial patients with sun-baths or electric-light baths, in addition to quinine.*

I have no doubt that Dr. King's reputation, in addition to his interesting and ingenious argumentation, will give an impetus to clinical tests in malarial districts of his hypotheses, tests which it would be a very simple matter to bring about. My object in writing these notes will be attained if they contribute to the possibility being held in view, at such clinical investigations, *that daylight can increase the favorable effect of quinine preparations upon malarial patients.*

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SOME PATHOLOGICAL AND CLINICAL ASPECTS OF ACQUIRED (FALSE) DIVERTICULA OF THE INTESTINE.

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Only a few years ago acquired diverticula of the intestine were looked upon as pathological curiosities, but owing to the work of Klebs, Heschl, Hanau, Good, Hansemann, Fischer, and Graser, more attention has been paid to them, and gradually their importance in relation to other abdominal conditions is becoming known. Even at the present day the direct clinical bearing of this condition is difficult to estimate, as the collected facts are rather meagre.

According to the old classification, diverticula were divided into false and true. The former became identified with acquired, the latter with congenital. The distinction based on the anatomical construction of the walls of these diverticula lay at the bottom of the first classification, but now we know that this, in many instances, is no distinguishing feature. False diverticula were considered mucosal herniæ through the muscularis, their walls consisting of serosa, submucosa, and mucosa. True diverticula, on the other hand, were made up of all the layers of the bowel wall, and Meckel's diverticulum was practically the only representative of this class. Recent work has shown that diverticula similar in structure to the true may develop during the patient's lifetime, and, as they have nothing to do with an embryonic development or a congenital structure, they should not be called true diverticula.

Klebs, Edel, Sudsuki, and others have reported acquired diverticula not due to direct traction on the intestine, in which all the layers of the intestinal wall were represented. In addition, Neumann, Nauwerck, and Hansemann have reported some cases due to direct traction by tumors (myoma, angioma) or by an accessory pancreas. These need not concern us, as they are exceedingly rare. Their production is clearly due to traction, and if they give any clinical picture, it will be due either to the primary disease or to some such pathological change as makes the diverticulum of Meckel give rise to symptoms. Those diverticula of all the layers of the gut wall not due to traction are often called true diverticula, as they differ from the true in no essential detail, as far as their intrinsic structure is concerned. They differ, however, in that they are acquired, and in this respect they and the more common mucosal herniæ above mentioned are grouped together. Both are acquired and both often develop in the same intestine. At times diverticula, with all the layers of the intestinal wall, develop first, and in them

the mucosa is pushed between the muscular bundles and a mucosal hernia results. From all this the distinction, based upon the time in which the diverticula develop, whether congenital or acquired, will surely appear simpler and lead to less confusion.

The acquired diverticula are found in the small intestine, in the appendix, in the colon, sigmoid flexure, and rectum. They are often multiple. At times hundreds have been found in one intestinal tract. It is fairly well agreed that they are most frequently found in the sigmoid, and that, in general, they are much more common than was once thought. Graser found diverticula ten times in twenty-eight cases, and in an investigation carried on by Sudsuki fifteen cases out of forty showed diverticula in the sigmoid. In the small intestine they are rather regular in their position with relation to the circumference of that part of the bowel. Here they develop at the mesenteric attachment, perhaps a little to one side of it, and by their growth gradually separate the leaves of the mesentery. Rarely do they become larger than a walnut—usually they are the size of a large pea or a chestnut; at times microscopic in size (Graser). In the large bowel no such regularity obtains and diverticula are found going into the mesentery or attached to some other part of the circumference of the bowel. Very often they develop in the haustra alongside one of the longitudinal muscular bands, and frequently they grow into the appendices epiploicæ.

As far as their causation is concerned, a unanimity of opinion does not exist. It is generally granted that old people alone show them, and especially such old people as have suffered from long-standing constipation. Klebs finds them in fat people, whereas Hansemann almost emphasizes the leanness of his subject. The former thought they were due to traction exercised on the bowel by the mesentery, whereas Hansemann favors the theory of pulsion from within the bowel. It is evident that Klebs' view would not explain those cases that had diverticula on the side opposite the attached mesentery—traction on the mesenteric side could hardly lead to the development of diverticula on the opposed side. Klebs pointed out that the diverticula on the mesenteric side were in close relation to the vessels of the bowel, and this has been verified by Hansemann and Fischer. On this point there is no doubt.

Heschl, Hanau, and Good, to find the cause of these diverticula, experimented by filling the intestines of human corpses with water and noting where the injected bowels ruptured. They were pleased to find that the rupture occurred regularly into the mesentery, and from this they concluded that here was the weakest part of the bowel; consequently just here diverticula developed. Hansemann went a step farther, verifying what had been "proven"—*i. e.*, the existence at the mesentery of a locus minoris resistentiæ, and, in addition, producing artificially false diverticula in the intestines of older people. These diverticula, he reports, ran along the sheaths

of the veins of the intestine and were similar, anatomically, to those produced by nature during the life of the patients.

Even if we could accept all this work as directly applicable to living conditions, we would still have to explain those diverticula that do not grow into the mesentery. *Why should these be produced by other agents than those leading to the mesenteric diverticula? Why should not all old people have diverticula, provided their bowels have been constipated, for surely they all have similar venous supply?* These investigators have not explained the origin of these structures, and only the anatomical part of their work is reliable.

A priori, one might have supposed that intestines subjected to increasing internal pressure would rupture at the mesentery. Here the bloodvessels normally enter and the many holes or spaces between the muscle bundles are filled during life with tense vessels containing blood. In the dead intestine altogether different conditions obtain. These spaces are all empty and non-resistant to internal pressure, the whole mesenteric wall of the gut being more in the nature of a sieve than of normal intestinal wall. Moreover, the distention caused by the injection of a stream of water resembles but very little the distention of the bowel within the body. In a recent article by Philipowicz the difference between the distensibility of the normal and dead bowel is emphasized. He never succeeded in distending the bowel to nearly the size it had while distended in the body from volvulus. The bowel ruptured long before an equal distention was attained. Chlumsky's experiments on the intestines of dogs throw some light on this side of the question. When he injected small intestines that were still alive, not removed from the normal animal, the rupture occurred opposite the mesentery, whereas, as soon as he worked with intestines removed from the animal and dead some ten or more hours, they ruptured into the mesentery (Experiment 7). In his earlier experiments with conditions approximating the normal, the ruptures never took place into the mesentery (Experiments 1 to 6).

In our experiments the process is peracute, whereas in the life of the individual it is very slow and chronic. If these facts were not enough to invalidate the experiments that have been published, and which I have been able to verify in greater part, the fact that clinically such intramesenteric ruptures are most exceptional, if they ever occur, shows that this is not the weakest part of the gut during life, as the above investigators declared. Frequently, in cases of ileus, the peritoneum and underlying muscle layers tear and separate, not at the mesentery, but usually more or less opposite to its attachment.

Chlumsky has demonstrated the same in the thicker intestines of dogs. First the serosa tears, then the muscularis, and, interesting to say, the circular first, later the longitudinal, and last of all the strong submucosa. All this shows that the weakest portion of the

gut wall is rather at some point on the side opposite the mesenteric attachment, rather than where Hanau, Heschl, Good, and others have placed it.

In studying this question, I proceeded as other investigators have done, and obtained similar results. The intestine, especially the small, ruptured almost regularly between the leaves of the mesentery, but now and again I obtained a rupture on the side opposite the mesentery. In the large bowel, in general, I had the same results, but here the ruptures were more often on the side where no mesentery was attached than in the small gut. The artificial diverticula reported by Hansemann I could not produce.

From all that has just been said, it will be seen that these experiments are valueless, because they do not show the weakest part of the living intestinal wall. Conclusions based on them, as to the origin of false diverticula, naturally must be invalid. Even if we granted that they were allowable, in trying to explain those diverticula that developed on the non-mesenteric side we would again be at a loss. But without going so far, it must be emphasized that the *supposed weakness at the mesentery does not exist, and, as it does not exist, it has nothing to do with the origin of acquired diverticula.*

In 1898 Graser added to our information on this subject, and tried, not without opposition, to connect chronic congestion of the bowel with the formation of the diverticula. A great deal of doubt has been thrown on this by the fact that congestion is not found in every case of diverticula. When the veins of the bowel are overfilled with blood, they must make more room for themselves and put aside the adjacent muscular fibres. This leads to the production of points of less resistance; in fact, almost reproduces the condition that we find when experimenting on the dead bowel. The enlarged spaces about the veins in which the blood is at a lower pressure, or the similar spaces more or less empty when the congestive condition is relieved temporarily by an increased arterial pressure, gives us a sieve-like condition of the mesenteric wall. Whereas Hansemann limited his application of the relation between veins and diverticula to mesenteric diverticula, Graser sees no necessity for such limitation and finds this relation between diverticula and veins, even in the non-mesenteric diverticula, as they occur in the sigmoid flexure. This view of Graser does not explain the diverticula that are not simple mucosæ hernial, but consist of all the layers of the gut wall; and the recent work of Sudsuki can be considered as a fairly conclusive overthrow of the congestion aspect of the question, though in some cases this may be a secondary factor in the development of diverticula. Not satisfied with disproving the general applicability of this theory, by demonstrating that only six out of his fifteen cases of acquired diverticula showed a condition of congestion and a cause for such a condition, Sudsuki, still clinging, more or less, to the older anatomical findings, propounded another theory. He regards a

diminished resistance of the connective tissue about the veins of the bowel as the predisposing cause of this condition, but he also leaves the etiological factors of the non-mesenteric diverticula more or less in the dark, and does not by this theory give any adequate explanation of the diverticula with muscular layers in their walls (of which he reported six instances), which naturally cannot be due to changes in the connective tissue in the vascular spaces, for the whole wall does give way.

No one can deny, after studying acquired diverticula, that the veins and their sheaths have a definite influence on the development of diverticula on the mesenteric side of the intestine, but from the fact that these diverticula often develop on the non-mesenteric sides, where no large veins pierce the bowel wall, it is more than likely that those on the mesenteric side do no more than direct the path along which the diverticula develop. There must be a cause that underlies both the mesenteric and the non-mesenteric diverticula, both those that are made up of serosa, submucosa, and mucosa, and those with these plus the muscular layers. From the similarity of the anatomical picture, it is highly probable that one and the same cause is present, no matter where the diverticula develop. Simple pulsion, due to collections of gas and feces, is not sufficient. If these alone sufficed, all people so afflicted would have diverticula, which is not the case. There must be some change in the resistant powers of the gut wall. The fact that *diverticula occur in old people, in people whose intestines have been more or less worked out, as evidenced by constipation, points to a muscular deficiency, and in this muscular weakness*, I think, *the cause of the formation of false diverticula must be sought.* This diminished muscular strength will explain the production of diverticula in the mesenteric and non-mesenteric parts of the bowel, for in those patients where the repeated increases in internal pressure overcome the diminished muscular strength of the wall, diverticula must result, diverticula of the whole gut wall or of the mucosa, submucosa, and serosa. According to this view, the latter or common form would develop when the muscular weakness is localized to a small area of muscular tissue, and, as in the mesenteric diverticula, the mucosa would be most affected and pushed along the lines of least resistance. The weak muscle bundles would give and be pushed slightly to either side, as is seen in many early and very small diverticula. The numerous vessels on the mesenteric side, especially the veins, would probably point the further path, and the intimate relation to the veins, which obtains in mesenteric diverticula, would be developed. When larger areas of the wall have lost their muscular strength, naturally all the layers of the wall will be found in the diverticula. Moreover, those diverticula with only longitudinal layers of muscle cells in their walls are equally well explained by this view of the process (Chlumsky). It is very probable that the original view of Klebs is, in part, correct, especially

with reference to the production of the diverticula in the small intestine, and that traction on the gut by the mesentery is a factor in the production of the necessary weakness at this point, where these diverticula so regularly occur in the small intestine. That this traction is a reality can be easily demonstrated by distending loops of intestine *in situ* or by noting the enormous tension of the mesentery in ileus cases. Naturally, in those parts of the intestine where no mesentery exists, this cannot be a factor.

The advantage of looking at this process in this way is manifold. It explains the production of all diverticula, no matter whether they are mesenteric or non-mesenteric in position, and no matter what their structure. It does not depreciate the changes that must develop in the muscular strength of an intestinal wall that has been overworked for a long time in emptying a bowel which is more or less in the position of a chronic ileus, due, perhaps, to torpidity of the rectum, without the ability to re-establish an equilibrium by adequate hypertrophy, not to mention the influence of old age. Moreover, it is in perfect consonance with experimental work and clinical observation.

So much for the causation of acquired intestinal diverticula. Before describing those changes in the diverticula which lead to serious consequences and give a more or less definite clinical picture, two facts must be emphasized. Diverticula in the large intestine, especially in the sigmoid flexure and rectum, are peculiar in that they alone are likely to undergo extensive pathological changes. Why diverticula in the small intestine are comparatively harmless cannot be definitely stated at the present moment. Judging, however, from the fact that in the large intestine hardened fecal masses which are lodged in the diverticula probably lead to the further changes, it can be said that the fluid consistency of the contents of the small intestine will probably account for the relative immunity of that part of the bowel from secondary pathological changes. In addition to this constant immunity of the small intestine, it must be emphasized that it is rather the exception than the rule for diverticula of the sigmoid and rectum to give rise to any symptoms. This probably explains the fact that these diverticula have received so little attention from clinicians. In the literature of this subject there are at least eighteen instances of inflammatory processes arising in these diverticula and leading to grave, often fatal, results.

If the mucous membrane of one of these diverticula in the sigmoid be examined, one can readily see an atrophic condition, in which both the epithelium and their glands are altered. Even before ulceration takes place there is visible an infiltration of round cells in the submucosa. As these diverticula are very often filled with hard fecal masses, ulceration of the mucosa, due perhaps to pressure, is liable to result. Such ulceration may lead to changes, both in the adjacent wall of the intestine, to similar inflammatory changes in the

mesentery of the sigmoid, also to similar changes about the bowel. Moreover, should such an ulceration develop rapidly, a perforation into the free peritoneal cavity results. If, however, such an ulceration should lead to the formation of adhesions between the sigmoid and another hollow organ, the perforation will lead to an anastomosis between the intestine and adherent viscus. At times such a diverticulum ulcerates into the mesentery and an abscess is formed between its leaves. At other times such an abscess may form in the wall of the gut or about it, and this may perforate into a hollow viscus, thus establishing a connection between intestine and bladder, this being the organ usually involved by a fistulous tract.

When the inflammatory process about the diverticula in the wall of the intestine has led to marked connective-tissue growth, this, by its secondary cicatricial contraction, occludes the lumen of the bowel more or less completely. Rotter has brought out the fact that these stenoses of the sigmoid and rectum differ, very markedly, from stenoses due to other causes. These are peculiar, in the fact that they are produced not by cicatricial processes in the mucosa and submucosa, but by the chronic inflammatory process in the deeper layers of the gut wall. In addition to this form of stenosis, diverticula lead to stenosis, both as a result of inflammatory changes in the mesentery and perisigmoid adhesions. Both of these are usually combined with the first form described. Perhaps the ileus cases described by Riedel as due to sigmoid mesenteritis are, in some cases, the result of thickening in the mesentery and angular bending of the intestine, caused by primary changes in acquired diverticula.

How often the carcinomata of the sigmoid arise from the chronic ulcerations of one of these diverticula is hard to determine, because so little attention has been paid to these structures. That such a tumor can arise from the mucous membrane of such a diverticulum, a case reported by Hochenegg conclusively proves.

False diverticula in the appendix have been reported by Kelynak, Ribbert, Edel, Fischer, and Mertens. Whether the earlier cases are acquired diverticula is doubtful. In a recent article by Mertens, it has been definitely shown, however, that diverticula are found in this organ, and that, here also, they may prove of very serious consequence. He reports ulceration and perforation of one of these, corresponding with a similar case of perforation of the diverticulum in the sigmoid, which he also reported.

From this short sketch of the pathological conditions that have been found in the eighteen reported cases, it is evident that acquired diverticula are not always as harmless as we are wont to regard them.

As far as the clinical picture is concerned, it, perhaps, would be best to group the reported cases according to the pathological condition found at operation or post-mortem, rather than according to the diagnosis.

1. *Diverticula which Produced Stenosis of the Sigmoid or Upper Rectum.* Of these I have been able to find six instances in the literature. Graser, Fabris, Rotter, Koch, Waldvogel, and Georgi have reported these cases. All were not simple cases of stenosis; for instance, Georgi's case was complicated by an abscess; Waldvogel's by a fistulous tract between the sigmoid and the bladder. Rotter's case shows, in addition to the stenosis, an abscess in the mesentery of the sigmoid, which communicated with the lumen of the intestine through six perforated diverticula. Koch reported a case of Rotter, which also showed perisigmoiditis, with a fistulous tract running to the bladder wall. All these cases suggested the diagnosis of carcinoma of the sigmoid (Rotter's, of the cæcum) leading to intestinal obstruction. The exact condition of affairs was not evident until the involved intestine was examined in detail; then the condition which had seemed, both clinically and at the autopsy, and *in vivo* during the operation, malignant, proved to be nothing but an inflammatory change arising from an acquired diverticulum.

From this it is evident that the prognosis of chronic ileus, due to sigmoid stenosis, even in the presence of a tumor (*e. g.*, Koch's case, where a movable mass was felt in the left iliac fossa; Georgi's case, where a nodular, sharply defined, fixed mass occupied the left iliac fossa; Rotter's case, with a mass as big as a child's head, etc.) must be very guarded. A colostomy above such a stenosis naturally would relieve the patient of the obstructive symptoms, and, perhaps, some of the good results in cases of supposed carcinoma of the sigmoid that have lived for years after the colostomy are just such cases as the above.

2. *Diverticula which Lead to Perforation into the Peritoneum.* In 1870 Loomis reported a case of peritonitis due to infection from a false diverticulum, which, however, showed no sign of rupture. In Georgi's article three other cases are mentioned, two reported by Fiedler in 1868, and one by Hilbert; all three cases had acute perforative peritonitis. Mertens reports a fourth case of peritonitis due to acquired diverticula, which had perforated into the free abdominal cavity. In this case a man, aged forty-five years, who had been constipated on and off, suddenly, after straining, developed a perforative peritonitis. At the operation the origin of the peritonitis was not found, and the patient died.

These cases teach a very important lesson. The sigmoid must be examined in cases of perforative peritonitis when the surgeon is in doubt as to the source of infection.

3. *Diverticula that Lead to Abscesses or Localized Peritonitis in the Left Iliac Fossa.* Georgi's case is an excellent instance of diverticula leading to suppurative processes in the left iliac fossa. Also in his case, which was complicated (as said before under 1) by stenosis, the probable diagnosis of carcinoma was made. This patient died of peritonitis following an exploratory laparotomy, at which the

tumor was declared inoperable. Post-mortem examination showed that the peritonitis owed its origin to the rupture of a suppurative focus about the sigmoid flexure and in its wall. That the tumor, instead of being malignant, was purely inflammatory, due to acquired diverticula. Gussenbauer, according to Mertens, had, many years ago, a similar supposed case of carcinoma of the sigmoid. The local peritonitis, in fact the whole tumor, when the resected gut was examined proved to be inflammatory, due to changes in acquired diverticula.

Such cases make it very clear that we must think of false diverticula when we are brought face to face with inflammatory conditions in the left iliac fossa in elderly people.

4. *Diverticula Leading to Perforation into the Urinary Bladder or Densely Adherent to the Bladder.* These and the stenotic cases are perhaps the most important from a prognostic standpoint. Abnormal communication between the bowel and the bladder suggests usually the gravest of diseases and an inoperable condition. Most of these cases that are due to carcinomata probably never get into the literature, so that a statistical table, such as Chavannaz has published, is of doubtful value. Most surgeons and pathologists, undoubtedly, see such communications most commonly after carcinoma of the bowel. Mikulicz speaks of such as if that were his experience. Still, the fact that in twenty such cases of communication between the bowel and bladder Chavannaz found that nine were inflammatory in origin, five tubercular, four carcinomatous, and two traumatic, is of great interest to us in connection with the question of diverticula. How many of the inflammatory cases were due to perforation of these into the bladder is not known, though the following cases will show that this has repeatedly happened, and that such a possibility must be reckoned with in these bowel-bladder fistulæ.

In the literature I have been able to find several cases of perforation into the bladder due to acquired diverticula. In 1858 Sidney Jones, in the *Transactions of the London Pathological Society*, reports the case of a man who passed feces and gas in his urine, and, finally, died, as the result of difficulty in urinating, because of a fecal concretion in the bladder. At the post-mortem examination a free communication was found between the bladder and a perforated sigmoid diverticulum. According to Waldvogel, Czerny had a somewhat similar case. Waldvogel himself saw a case in which the patient, who had been passing gas in his urine, had a communication between the bowel and bladder by a sinuous fistula. From the fact that the gas was odorless and contained no sulphur, he concluded that it was produced in the bladder. He was able to isolate a gas-producing bacterium, which he thinks wandered in from the intestine by the fistulous tract. In Mertens' article another case attributed to Treves is reported. Koch's case, of which mention has already

been made, under stenosis, showed intimate adhesions to the bladder wall, so that part of this had to be resected. This patient had urinary symptoms, and the natural suspicion before operation was primary carcinoma of the gut, with secondary involvement of the bladder.

Hepner recently reported another case in an old woman. The symptoms came on suddenly after lifting a heavy weight. She had fever and pain in the abdomen and vomiting. Gradually the urine became turbid, and before long she showed all the typical signs of an anastomosis between the bladder and sigmoid. The fistulous tract was tied off by operation, and the condition was clearly shown to be a sigmoid diverticulum which had opened into the bladder.

5. *Diverticula and Mesenteritis.* Most of the cases mentioned have shown more or less marked thickening and contraction of the mesentery. As said before, it still remains to be proven that such changes due to acquired diverticula lead to that form of chronic ileus which Riedel cures by non-operative means. Theoretically, this relation seems plausible, and in a recent article by Brehm this author tries to establish a definite relation between inflammation in diverticula, mesenteritis, and volvulus. In eleven out of twenty cases of volvulus he found well-marked mesenteric changes, but he did not prove the first link in the chain. Perhaps in time that will be made clear.

6. *Diverticula and Appendicitis.* Mertens has given most time to the study of this relation. He found, in 106 appendices taken from corpses, two instances of diverticula, and in twenty-eight diseased appendices removed by operation he found one diverticulum which had perforated during the attack. As the appendix itself is really nothing but a diverticulum and subject so frequently to disease, it is a little doubtful whether false diverticula in this location are of much moment in bringing on the trouble. Prognostically, of course, they are of little or no importance, in comparison with those cases in and about the sigmoid that simulate malignant growths.

7. *Diverticula and Carcinoma.* This relation is shown by just one case, that of Hochenegg (Thirty-first Congress of German Surgeons). The future alone can show how important in the production of carcinomata the chronic ulcerations in false diverticula are.

Whether such cases of intra-abdominal disease as I have just enumerated, due to acquired intestinal diverticula, will be recognized more frequently in years to come, it is difficult to say. Only twenty years ago Virchow spoke of acquired diverticula as an unusual pathological condition, but during these twenty years our views have changed. Perhaps the same will occur in the study of the intra-abdominal conditions due to false diverticula, and before long many cases like the eighteen here gathered will be reported. Then will we be in a better position, I trust, to differentiate between the inflammatory processes due to intestinal diverticula and those malignant diseases for which they are mistaken. Already we

are in a position to profit from the reported cases, both in our treatment and in our prognosis.

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THE COMPLICATIONS OF AMŒBIC AND SPECIFIC DYSENTERY, AS OBSERVED AT AUTOPSY.[1]

AN ANALYSIS OF ONE HUNDRED AND TWENTY CASES.

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IN the study of post-mortems on patients dying from dysentery at the General Hospital, I have been impressed with the frequent occurrence of grave complications in this disease, and have paid special attention to the pathological conditions present in the various organs. These conditions have been so constant and so important that it has seemed to me that they should be considered more at length than they are now in most of the text-books upon medicine. In looking up the subject of the complications of dysentery, in the most recent works, including even those of the larger

[1] With permission of the Surgeon-General of the United States Army. Read at the February, 1904, meeting of the San Francisco County Medical Society.

systems, I have been surprised at the relatively scant attention which this subject has received. For instance, in Davidson's excellent article upon dysentery in the second volume of Allbutt's *System of Medicine*, no mention whatever is made of the complications attending the disease. In La Fleur's article upon amœbic dysentery in the same volume, the complications attending it are dismissed in a very few lines, the only ones receiving much attention being abscess of the liver and intestinal perforation. This lack of extensive consideration of the complications of this disease is found in almost all of the more exhaustive works upon medicine. To the author this is surprising, for the reason that in a majority of cases occurring at the General Hospital, death has resulted rather from some complication than from the disease itself. While it is possible, in the class of cases cited, that many of the complications may have been due to campaigning in tropical countries with the incident hardships and lack of hygienic living, yet many of them are so constant that it is impossible not to believe that they are due to the disease, rather than to other factors.

It will be noticed that I have divided dysentery into two forms, amœbic and specific. This division has been made because the clinical and pathological conditions present are so diverse as, in my mind, to clearly separate them. Under the term "amœbic dysentery" is included the dysentery caused by the infection of the intestinal tract with *amœbæ coli*. Under the term "specific dysentery" I include the chronic form of the acute epidemic dysentery due to the bacillus of Shiga. This specific form of dysentery is differentiated from the amœbic form by the absence of amœbæ in the feces, by the occurrence, in a majority of cases, of agglutination of a pure culture of the Shiga bacillus by the blood serum of the patient suffering from the disease, and by the isolation of this bacillus, in pure culture, from the feces or from the intestinal ulcerations.

In a previous communication[1] I have described in detail the pathology of the chronic specific form of dysentery as observed in cases dying from this disease, and have been able, in several cases, to separate from the intestinal ulcerations the Shiga bacillus in pure culture. Although the differentiation of this form of dysentery by means of the agglutination test is not, I believe, entirely reliable, still I may say that in my experience this test has proven of much value. I have made a large number of tests with the Shiga bacillus in cases both of amœbic and specific dysentery, and have been able, in a majority of cases, to separate the two by this method. However, we do not have to depend upon this test in order to distinguish the two varieties, as the finding of the amœbæ in the

[1] The Pathology of Chronic Specific Dysentery of Tropical Origin, American Medicine, October 11, 1902.

feces decides at once whether or no we are dealing with amœbic dysentery.

When, however, the cases come to the autopsy table, the pathology of the two forms of dysentery are so different that there can be no mistake made in deciding with which we have to deal. It is not my intention here to give a description of the pathology of the various forms of dysentery, but simply to say that the pathological conditions present in the intestines in amœbic dysentery are so entirely distinct from those present in the specific form that a glance at the intestine suffices to distinguish them.

In considering the complications of these two forms of dysentery, I have selected for analysis 120 cases, 60 of the amœbic form and 60 of the chronic specific form. I have taken an equal number of cases of both varieties for the sake of comparison. I wish to state that the 60 cases of amœbic dysentery comprise nearly all the deaths we have had from that variety, and it is an interesting fact that the chronic specific form was very much more numerous at this hospital during the early days of the Philippine insurrection than it has ever been since. This agrees very well with the fact that the acute epidemic dysentery (or specific dysentery), as studied by Flexner and Strong, was very prevalent in the Philippines during the early days of the insurrection, but has since nearly disappeared, as I am informed by medical officers who have recently returned from the Islands. That this is true is proven by the fact that almost all of the cases of dysentery now received at the General Hospital are cases of the amœbic variety, whereas during 1899 and 1900 the vast majority of the cases received were of the chronic specific form.

Complications Observed in the Nervous System.

The cerebrum, in nearly every case in which it was examined, showed an increase in the amount of cerebrospinal fluid. This increase was often excessive, the brain being œdematous. Upon section of the brain, the lateral ventricles were invariably found filled with fluid, oftentimes slightly cloudy and the choroid plexus was invariably congested. A peculiar condition noted in all the amœbic cases was the intense congestion of the bloodvessels of the cerebrum. This congestion was very marked in all cases examined, and in about 50 per cent. of the cases minute capillary hemorrhages had occurred, which were scattered irregularly over the surface of the cerebrum; in the cases of chronic specific dysentery, on the other hand, the surface of the brain appeared anæmic, and this was especially true of the cut surface. In 3 cases of amœbic dysentery, and in 2 of the gangrenous stage of the chronic specific form, small areas of softening were noticed in the frontal lobes near the median fissure.

Complications Observed in the Respiratory System.

The most common conditions found complicating dysentery in the lungs were emphysema and bronchopneumonia. Of the 120 cases, 62 showed an emphysematous condition of the lungs, of which 42 occurred in the chronic specific form, the remainder in the amœbic. This condition is probably due, in greater part, to the long-sustained recumbent position of the patient and to the poor circulation which is generally present in these cases.

Bronchopneumonia is the most important lung complication, occurring in 14 cases, 12 of which were of the chronic specific form, and 2 of the amœbic. Perhaps a better name to designate this form of pneumonia is hypostatic, as it is probably due to maintaining the recumbent position for a long period of time, but as the pathological conditions are identical with those found in bronchopneumonia, I have preferred to retain that name. This complication was invariably fatal.

Lobar pneumonia occurred in 9 cases, 7 in the chronic specific form, and 2 in the amœbic. In all the cases it was the cause of death.

Empyema occurred in 11 cases, 10 of them being in the amœbic form of dysentery, and 1 in the chronic specific form. Of the 10 cases occurring in amœbic dysentery, 8 were due to perforation of a liver abscess into the pleural cavity.

A very common condition found at autopsy in both forms of dysentery is a chronic adhesive pleurisy, which occurred in 26 of the cases—19 of the specific and 7 of the amœbic form; in all of these cases the adhesions binding the lungs to the chest walls were very firm and of long duration.

Chronic bronchitis occurred in 16 cases—in 10 of the specific and in 6 of the amœbic form. Acute bronchitis occurred in 2 cases of amœbic dysentery. Acute pleurisy occurred in 8 cases, 6 of them being of the chronic specific form and 2 of the amœbic. Infarct of the lung occurred in 1 case of the chronic specific variety. Pulmonary tuberculosis complicated both forms of dysentery, 10 cases showing this complication; 6 of them of the amœbic variety, and 4 of the chronic specific form. In 6 of the cases death was due to this complication. Abscess of the lung was found in 4 cases; in 2 of the cases the abscesses were very numerous, being metastatic in character, and occurred in the chronic specific form of the disease. In 1 of these cases metastatic abscesses of the kidney were also found. The remaining 2 cases were due to amœbic infection of the lung, in 1 case the abscess being due to the perforation into the lung tissue of an amœbic abscess of the liver, and in the other to primary infection of the lung by amœbæ, a rather rare condition. In nearly every case a chronic venous congestion of the lungs was observed, but I have considered this of sufficient importance to record in but 59 cases. This congestion was, of

course, due to the recumbent position of the patient for a considerable period of time, and in many instances led to the production of a bronchopneumonia. I have only considered the condition as one of bronchopneumonia when the characteristic pathological lesions were present upon microscopic examination of sections of the lung, and I may say here that in all the cases recorded careful microscopic examinations were made of all the viscera, besides the examination of the organs macroscopically.

From a consideration, then, of the complications occurring in the respiratory system in dysentery, it will be seen that the most dangerous are the varieties of pneumonia. It will also be noted that the chronic specific variety of the disease is more often accompanied by serious complications than is the amœbic form. This is especially true of the occurrence of pneumonia, and is, no doubt, due to the fact that in the chronic specific variety the patient is more often compelled to keep to his bed for a longer period of time than when he is suffering from amœbic infection.

Complications Observed in the Circulatory System.

Œdema of the pericardium occurred in 30 cases—in 18 of the chronic specific form, and in 12 of the amœbic form. This condition I have found to be one of the most characteristic complications observed in the fatal cases, and, as a rule, the pericardial cavity was found nearly filled with a clear, straw-colored fluid, although in a few cases small shreds of fibrin were observed. Both layers of the pericardium appeared normal so far as could be judged. Acute pericarditis occurred in 6 cases—in 4 of the chronic specific variety, and in 2 of the amœbic. In both instances found occurring in amœbic infection, the condition was due to the perforation of an amœbic abscess of the liver into the pericardial cavity. In all the cases of pericarditis the effusion was purulent in character and the pericardial cavity was distended by it. Valvular disease of the heart was present in 1 case suffering from the amœbic form of the disease. In this case there was valvular disease of both aortic and mitral valves. Arteriosclerosis was observed in 12 cases —8 suffering from the chronic specific form of dysentery, and 4 from the amœbic form. It is impossible in these cases to say how much alcohol had to do with the sclerosis, although it is reasonable to suppose that in most cases it had some etiological influence. When we take into consideration, however, the fibrosis which occurs in dysentery, especially in the liver, spleen, and kidney, it is impossible not to believe that the arteriosclerosis may be partially due to this general hypertrophy of connective tissue.

An important condition found microscopically in the heart muscle, especially in the chronic specific form of dysentery, is a

brown atrophy of the muscle fibre accompanied by partial fragmentation. This condition, the nature of which is still a disputed point, can be observed in almost every case if careful sections are made from different portions of the heart muscle, and as it occurs so frequently in dysentery, it is, I believe, due to pathological changes in the muscle fibre. This atrophy results naturally in lessened tonicity of the heart muscle, and, therefore, in lessened circulation, thus favoring the chronic venous congestion of the organs, which is so common in both varieties of the disease.

Complications Observed in the Genito-urinary System.

Kidneys. Some pathological condition of the kidneys was observed in almost every case of dysentery which has come to autopsy in this hospital. Thus, of the 120 cases analyzed in this paper, 101 showed some form of nephritis. It must be remembered that I have not diagnosed nephritis in these cases alone from a macroscopic examination of the organs, but also from a careful microscopic examination. There is no complication occurring in dysentery which is so common and, I believe, so important from a practical standpoint as is the occurrence of nephritis. It has been stated by some authorities that nephritis occurs but rarely in dysentery. I cannot understand how such a statement can be made, unless the cases which we receive from the Philippines are exceptional in some way, and this I do not believe. It seems to me that the statement can only be made by those who have had very little experience with the disease. Of the thousands of cases of dysentery which have passed through the General Hospital at San Francisco, I am prepared to state that at least 60 per cent. showed at some time or other evidences of inflammation of the kidney. The urine of at least this proportion showed, at some time or other, albumin and casts, and while the condition in a vast majority of cases seemed to clear up, we must admit that during a certain period of time some form of nephritis was present. This very large percentage is well shown in the fatal cases occurring at the General Hospital, in which 101 presented well-marked macroscopic and microscopic evidences of nephritis in one of its forms. The 101 cases were divided, as regards the type of nephritis, as follows:

Acute parenchymatous nephritis	36
Subacute parenchymatous nephritis	45
Chronic interstitial nephritis	20

As regards the occurrence of these various forms in the two varieties of the disease, they were divided as follows:

Chronic specific form:	Acute parenchymatous nephritis	19
	Subacute parenchymatous nephritis	23
	Chronic interstitial nephritis	6—48
Amœbic form:	Acute parenchymatous nephritis	17
	Subacute parenchymatous nephritis	22
	Chronic interstitial nephritis	14—53

It will be observed that a greater number of cases occurred in the amœbic variety than in the chronic specific. This is an exception to the general rule that complications of other organs, except abscess of the liver, are more frequent in the chronic specific variety of the disease than in the amœbic. This large percentage of cases of dysentery showing marked kidney lesions would seem to indicate that the kidneys are especially susceptible to the poisons which are elaborated during the process of the dysenteric infection. As to the nature of these poisons, we know absolutely nothing; but so far as a careful examination could detect, there was nothing peculiar noticed about the microscopic appearance of the sections of the kidneys, nor could there be detected any accumulation of micro-organisms, nor any evidence of any special form of cellular degeneration. It is interesting to note that chronic interstitial nephritis occurred in 20 cases, because in all these cases the patients were young men in whom such a condition would not be expected. Of course, the question of alcoholism has to be considered in these cases, but I do not believe it had very much to do with the production of the disease, and think rather that the condition was one of overgrowth of connective tissue stimulated by some poisonous material, elaborated during the progress of the disease. The occurrence of so many cases of nephritis complicating the dysenteric process, indicates how important an examination of the urine is, since the non-discovery of this complication will invariably lead to a much less favorable prognosis as regards the course of the dysentery, as treatment will not be directed toward the kidneys.

It is impossible for me to say how many of these cases died from the effects of the nephritis, but I believe a considerable proportion did so, and in many of them the dysenteric lesions were so nearly healed that we were forced to look for the cause of death in some other organ, and I believe the kidney was, in all these cases, the organ at fault. Certainly 101 cases of nephritis occurring in 120 fatal cases of dysentery, which were not selected to show this complication, is very significant, and it seems to me of great practical importance.

Infarct of the kidney was observed in 1 case of amœbic dysentery. Abscess of the kidney was observed in 2 cases; in 1 the abscess being due to a perforation of an amœbic abscess of the liver into the kidney, and in the other it was due to metastatic abscesses, which were found also in the spleen and lungs. Urinary calculi were observed in 1 case of amœbic dysentery. Pyelonephritis was observed in 2 cases; 1 of the amœbic form, and the other of the chronic specific form.

Complications Observed in the Glandular System.

Liver. The most common complication observed in the liver was fatty degeneration, which was present in 42 cases, of which

22 were suffering from chronic specific dysentery and 20 from amœbic. In most of the cases microscopic examination showed the degeneration to be very extensive, whole lobules of the liver tissue being replaced by fat.

The next most frequent complication was chronic venous congestion, which was present in 36 cases, 20 of the chronic specific form and 16 of the amœbic. Microscopically this condition was often found to be very marked, the capillaries being engorged with blood, and in many instances these had ruptured, forming hemorrhagic areas within the organ.

Cirrhosis of the liver was observed in 10 cases, 6 of the chronic specific form and 4 of the amœbic. In all, the condition was one of common interlobular cirrhosis of comparatively slight extent. In those cases which did not present evidence of cirrhosis macroscopically, the microscope revealed what was evidently the earliest stage of the process, as evidenced by a slight increase in the interlobular connective tissue and more or less infiltration of the portal spaces by leukocytes and newly formed connective-tissue cells.

Abscess of the Liver. Undoubtedly the most important complication of dysentery, that is, of the amœbic form, is abscess of the liver. This condition was observed in 24 cases which came to autopsy, of which 22 were suffering from amœbic dysentery and 2 from the chronic specific form. The abscesses occurring in the chronic specific form were metastatic in character, the liver being thickly crowded with minute foci of suppuration.

The frequency of the occurrence of amœbic abscess of the liver in dysentery seems to vary greatly with different observers, as is well shown in the following table. The total number of cases of dysentery observed at autopsy or collected from autopsy records (with the exception of Councilman and La Fleur and Futcher, who give all cases observed) by the different investigators is given, followed by the percentage of those presenting abscess of the liver. All the cases given in the table as observed by myself were amœbic in character, the *amœba coli* being demonstrated in the feces.

Observers.	No. of cases observed.	Percentage.
Kartulis	Over 500	60
Zaucarol	444	59
Edwards and Waterman	699	72.1
Councilman and La Fleur	1429	21
Craig	74	33.7
Futcher	119	22.6
Smith	45	84.4

As will be seen from the above table, the percentage of cases of dysentery showing abscess of the liver at autopsy, as observed by myself, has been very small compared with that observed by others. I can hardly tell how to explain this difference unless most of the cases of amœbic abscess are discovered in the Philippines,

and either end fatally there or are operated upon before reaching this country, whereas the vast majority of cases of dysentery not presenting this complication are returned to this country for convalescence.

As regards the location of the abscesses, it may be stated that they occur most frequently in the right lobe, especially in the posterior and upper portion of that lobe. The following table, showing the location of the abscesses observed in cases coming to autopsy here, illustrates this point very clearly.

Number of case.	Abscesses.			
	Single.	Multiple.	Number.	Location.
1	.....	Yes	6	Right lobe 5, left lobe 1
2	.....	"	8	" " 8, " " 0
3	.....	"	4	" " 3, " " 1
4	.....	"	13	" " 8, " " 5
5	.....	"	8	" " 8, " " 0
6	Yes	.....	1	" " 0, " " 1
7	"	.....	1	" " 1, " " 0
8	.....	Yes	2	" " 2, " " 0
9	Yes	.....	1	Lobus Spigelii.
10	.....	Yes	17	Right lobe 16, left lobe 1
11	.....	"	too numerous to count	In both lobes.
12	.....	"	10	Right lobe 2, left lobe 8
13	Yes	.....	1	" " 1, " " 0
14	.....	Yes	3	" " 3, " " 0
15	.....	"	5	" " 5, " " 0
16	Yes	.....	1	" " 0, " " 1
17	.....	Yes	3	" " 3, " " 0
18	Yes	.....	1	" " 1, " " 0
19	"	.....	1	" " 1, " " 0
20	.....	Yes	very numerous	Right lobe, all but 2.
21	Yes	.....	1	" " 1, left lobe 0
22	.....	.....	30	" " 23, " " 7
23	.....	Yes	4	" " 4, " " 0
24	Yes	.....	1	" " 1, " " 0

In those cases which showed multiple abscess of the liver it was almost invariably found that the oldest abscesses, as shown by their size and the thickness of the abscess wall, occurred in the right lobe posteriorly or near the dome. In only 1 case of multiple abscesses observed did the pathological findings seem to indicate that the abscess formation commenced first in the left lobe. My experience, as given in the above table, is well borne out by the collective investigation made by Roux as to the location of liver abscesses in amœbic dysentery. He collected 639 cases with the following result: 435, or 70.8 per cent., were situated in the right lobe; 85, or 13.3 per cent., in the left lobe; and 2, or 0.3 per cent. in the lobus Spigelii. A study of my cases, as given in the table, will also show that the majority of the cases showed multiple abscess, thus proving that the general opinion that amœbic abscess is nearly always single is a mistake. In fact, some authorities have counted several thousand abscesses in the liver and have separated amœbæ from these abscesses. I have seen instances in which the abscesses were too numerous to count.

Rupture of a liver abscess is of comparatively frequent occurrence, the perforation taking place into the abdominal cavity, the pleura, the pericardium, or into any of the adjacent viscera. In 24 cases observed here, 7 were accompanied by rupture, perforation occurring in the following localities: Right pleural cavity, 5 cases; left pleural cavity and the pericardial cavity, 2 cases. As the location of the rupture of amœbic abscess is of great surgical importance, I have prepared the following table giving the results of various observers as regards this question.

Observers.	Case of liver abscess.	Case of rupture.	Pericardium.	Pleura.	Lung.	Colon.	Stomach.	Bile ducts.	Vena cava.	Kidney.	Lumbar region.
Waring . .	300	68	14	28	15	2	1	1	3	2	2
Dutroulau . .	66	25	2	10	7	1	1	...	...	...	4
Roux . . .	162	54	11	17	14	3	6	2			
Haspel . . .	25	6	4	2							
Cambay . .	10	3	...	2							
Howard . .	6	5	5								
Craig . . .	24	8	and pleura 2	5	...	...	...	...	...	1	

In one case observed at this hospital an amœbic abscess of the liver was operated upon successfully, in which no history of a previous attack of dysentery could be obtained. That an amœbic abscess can occur primarily in the liver without a previous dysentery, has been established beyond a doubt by a case reported by Buxton of a woman dying at the Philadelphia Hospital, in which the autopsy showed four large abscesses in the right lobe and one in the left lobe of the liver, the pus of each containing amœbæ, although the large and small intestines were absolutely normal in appearance. In all probability, the case which I have just mentioned is a similar one to that of Buxton.

It is not my intention here to discuss the etiology of amœbic abscesses further than to say that they are, without doubt, due to the infection of the liver tissue by the *amœba coli*, probably reaching the organ through the portal vein, although Councilman thinks that they reach the liver through the peritoneal cavity after penetrating the intestine. Other authorities regard the lymphatics as responsible for the liver infection.

Bacteriological Examination. In all the cases which came to autopsy, sections were made of the walls of the liver abscesses, and in the majority of cases amœbæ were demonstrated. An examination of the pus from the abscess cavity did not result, as a rule, in a demonstration of amœbæ unless it was removed by scraping from the abscess wall. By the later method amœbæ were demonstrated without difficulty. A bacteriological examination of the pus by means of cultures resulted in about 50 per cent. of the cases showing a mixed infection with some other organism, princi-

pally staphylococci, streptococci, or the *bacillus coli communis*. My observations as regards the bacteriology of the pus in liver abscess confirms that of many other observers who have found that in about one-half the cases there is a mixed infection. It is in these cases, also, that we find the yellow or greenish pus, rather than the reddish or chocolate-colored pus, which is so typical of amœbic infection.

The Spleen. Altogether the most common complication observed in the spleen was cirrhosis of that organ, which was present in 48 cases, 25 of the chronic specific form of dysentery and 23 of the amœbic form. Infarct of the spleen was observed in 7 cases, 5 of the chronic specific form and 2 of the amœbic form. A very common condition found in the spleen was pigmentary degeneration, which was especially noticeable in sections of the organ, the pigment being in the form of irregular collections of fine granules, brownish-yellow in color, scattered especially along the edges of the fibrous trabeculæ and around the Malpighian corpuscles. In 21 cases melanosis of the spleen was very noticeable, 16 of the cases suffering from amœbic dysentery. As, in almost every case the melanosis was due to previous malarial infection, it is of interest as showing how often amœbic dysentery is complicated by malarial fever.

Complications Observed in the Digestive System.

As would be expected, chronic enteritis is the most frequent of all complications in the digestive system. This condition was observed in 70 cases, 42 of the chronic specific form, and 38 of the amœbic. The enteritis present varied considerably, of course, as regards its extent and severity, but it may be stated as a general rule that all cases of dysentery are complicated by an enteritis of at least medium severity, and it is my belief that this enteritis is of very great importance as regards the prognosis as to recovery. Many cases which have died at this hospital have undoubtedly succumbed from the effect of the enteritis, rather than from the dysenteric infection.

Chronic gastritis is next in frequency to enteritis as a complication, occurring in 60 cases, 31 of the chronic specific form and 29 of the amœbic form of dysentery. In all the cases the gastritis present was of severe character, and taken with the enteritis certainly militated greatly against the patients' recovery.

Perforation of the intestine through an ulceration occurred in 4 cases, 3 of them suffering from amœbic dysentery, and 1 from the chronic specific form. All of these cases resulted in death from peritonitis.

Acute peritonitis complicated the dysenteric process in 10 cases, 8 suffering from the amœbic form and 2 from the chronic specific form; 4 of these cases were due to perforation by dysenteric ulcers, and 6 to pus infection from liver abscesses.

In this study of the complications of dysentery, I have tried to call attention to their great frequency and importance, as I believe they are often overlooked, much to the detriment of the patient's welfare. It will be seen that the chronic specific form of the disease is most prolific in complications, and in our experience this form of dysentery has been the most fatal with which we have had to deal. The most serious complications accompanying the disease, either specific or amœbic, are nephritis, abscess of the liver, and chronic enteritis and gastritis. These occurred so frequently, especially the two latter, that treatment should be especially directed toward them.

In conclusion, I append a table showing the complications observed in the 120 cases analyzed, which may prove of service to students of this subject.

Total number of cases 120: amœbic, 60; chronic specific, 60.

Complication.	Total number.		Amœbic dysentery.	Chronic specific dysentery.
Subacute parenchymatous nephritis	45	101	22	33
Acute parenchymatous nephritis	36		17	19
Chronic interstitial nephritis	20		14	6
Chronic enteritis	70		38	42
Chronic gastritis	60		29	31
Venous congestion of lungs	59		24	35
Cirrhosis of spleen	48		23	25
Fatty degeneration of liver	42		20	22
Venous congestion of liver	36		16	20
Chronic pleurisy	26		7	19
Abscess of liver	24		22	2
Melanosis of spleen	21		16	5
Chronic bronchitis	16		6	10
Œdema of pericardium	12		2	10
Empyema	11		10	1
Bronchopneumonia	14		2	12
Cirrhosis of liver	10		4	6
Arteriosclerosis	12		4	8
Pulmonary tuberculosis	10		6	4
Acute peritonitis	10		8	2
Acute pleurisy	8		2	6
Lobar pneumonia	9		2	7
Acute pericarditis	6		2	4
Infarct of spleen	7		2	5
Abscess of lung	4		2	2
Intestinal perforation	4		3	1
Acute bronchitis	2		2	
Pyelonephritis	2		1	1
Abscess of kidney	2		1	1
Biliary calculus	1		...	1
Infarct of lung	1		...	1
Infarct of kidney	1		1	
Urinary calculi	1		1	
Valvular disease of heart	1		1	

REVIEWS.

CLINICAL PATHOLOGY OF THE BLOOD. A TREATISE ON THE GENERAL PRINCIPLES AND SPECIAL APPLICATIONS OF HÆMATOLOGY. By JAMES EWING, A.M., M.D., Professor of Pathology in Cornell University Medical College, New York City. Second edition, revised and enlarged. New York and Philadelphia: Lea Brothers & Co.

MUCH knowledge and interest have been derived within the last few years from numerous hæmatological studies and from investigations upon the hæmatopoietic organs and histogenesis of blood cells, and to-day the importance which the pathology of the blood bears to scientific medicine in general can scarcely be overestimated. That there should be some ready means of access to the facts concerned in this rapidly developing subject is obvious, and inasmuch as the last two years represent a rapid advance in many divisions of the subject the second edition of Dr. Ewing's book becomes of immediate interest. The enlargement and revisions have succeeded in producing an excellent book. Many valuable additions have been made in the chapter on Technique, where new and useful apparatus is described; but it is rather surprising that no mention is made of Wright's stain, although the Jenner mixture is given. A description of cryoscopy and the serum test for blood form part of the new material in this portion of the text. A very brief account of Ehrlich's theories on immunity is inserted. Rather extensive and valuable additions have been made in the discussions on the morphology of the blood and in the chapter devoted to leukæmia, while in the appendix one finds a paragraph concerning the interesting condition termed by v. Leube "Leukanæmia." In fact, new material has been added in almost all parts of the volume, and in every place one finds mention made of the latest researches on the histology and pathology of the blood.

The bibliography appended to each chapter has been carefully brought up to date, and forms one of the most notable and excellent features of the volume. Some new plates and illustrations have been inserted in the text, but most of them are of the same general character as those in the first edition, and practically all of the old plates are used again. They do not give one a good idea of blood cells, and appear crudely diagrammatic. But, in spite of the illus-

trations, the volume is fundamentally good, and has so many really excellent qualities that as a text-book and treatise for reference it is almost invaluable. W. T. L.

THE FOUR EPOCHS OF WOMAN'S LIFE. By ANNA M. GALBRAITH, M.D. Philadelphia, New York and London: W. B. Saunders & Co., 1903.

WE do not believe that this book fills any need. In a former review the question of the necessity of books of this class was considered, and it does not seem needful to go over the same ground again. Suffice it to say that, while no criticism is called for regarding the statements made in the volume, we are old-fashioned enough to consider the young woman better off without the knowledge here offered.

We are glad to see that hemorrhage of the menopause receives in this edition considerable notice. If the book is to be read by the laity it is well that one of the most important subjects in the whole range of woman's reproductive life be not slighted, as was the case in the first edition of the work. W. R. N.

THE PRACTICE OF MEDICINE. A TEXT-BOOK FOR PRACTITIONERS AND STUDENTS, WITH SPECIAL REFERENCE TO DIAGNOSIS AND TREATMENT. By JAMES TYSON, M.D., Professor of Medicine in the University of Pennsylvania and Physician to the Hospital of the University; Physician to the Pennsylvania Hospital; Fellow of the College of Physicians of Philadelphia; Member of the Association of American Physicians, etc. Third edition, thoroughly revised and in parts rewritten. With 134 illustrations, including colored plates. Philadelphia: P. Blakiston's Son & Co., 1903.

IT is not surprising that a third edition of Dr. Tyson's Practice should be required, for the work is on the whole one of the most thoroughly satisfactory of the more recent treatises on that subject. In the new edition both the text and the illustrations have been somewhat added to, and the revisions are numerous and, for the most part, have been carefully made. A short, clear statement upon paratyphoid fever has been added. In the classification of types of dysentery a change is to be seen whereby "Diphtheritic Dysentery" is replaced by "Bacillary Dysentery."

The difficulties of classification are made evident by the fact that under Infectious Diseases the author has thought it wise to place not only croupous pneumonia, but also bronchopneumonia, chronic interstitial pneumonia, and embolic pneumonias.

In accordance with the prefatory request, we take the liberty of calling attention to a few errors of omission and commission. A curious typographical mistake which has gone through all three editions ascribes the investigation of hypertrophic cirrhosis to "the French clinicians, headed by Charcot and Henoch!" (page 460). Angina Ludovici is classed under Diseases of the Salivary Glands, and the statement is made that it is "an inflammation of the floor of the mouth, beginning in the submaxillary gland" (page 314).

It is somewhat surprising to find no mention either of multiple serositis or of adiposis dolorosa, in view of the considerable attention those affections have recently received.

The value of the book to physicians is much increased by the many references to the literature, while the clear descriptions and systematic arrangement make it admirably fitted for the use of advanced students. L. A. C.

MODERN METHODS IN THE SURGERY OF PARALYSES, WITH SPECIAL REFERENCE TO MUSCLE-GRAFTING, TENDON-TRANSPLANTATION, AND ARTHRODESIS. By A. H. TUBBY, M.S. LOND., F.R.C.S.E., Surgeon to and Lecturer on Clinical and Orthopedic Surgery, and in Charge of the Orthopedic Department at Westminster Hospital; Senior Surgeon to the Evelina Hospital for Sick Children; Surgeon to the National Orthopedic Hospital, etc.; and ROBERT JONES, F.R.C.S.E., Honorary Surgeon to the Royal Southern Hospital, Liverpool; Honorary Surgeon, Liverpool Country Hospital for the Chronic Diseases of Children, etc. London: MacMillan & Co., Limited. New York: The MacMillan Company, 1903.

PROBABLY in no department of orthopedic surgery has more progress been made than in the prevention and treatment of deformities resulting from paralysis, and a convenient, readable monograph setting forth this advance will be certainly very acceptable to the profession. The authors of the book are familiar to the American profession through their numerous contributions to surgical literature. They have not deemed it necessary to do more than mention the older and simpler methods of treating deformities resulting from paralysis, such as tenotomy and mechanical devices, as these are to be found in all standard works on orthopedic surgery. Attention is principally given to the methods of muscle-grafting, tendon-transplantation, and arthrodesis. The book is divided into three sections, the first dealing with Infantile Paralysis or Acute Anterior Poliomyelitis, the second with Infantile Spastic Paralysis, or Cerebral Palsy of Children, and the third with Paralysis and Deformities Arising from Injuries and Diseases of Nerves and Some Degenerations of the Spinal Cord.

Anyone reading this small volume and who has not followed the recent advances made in the treatment of these conditions will be surprised at what present-day surgery is able to accomplish.

The illustrations are not numerous and are usually diagrammatic, but are sufficient to thoroughly elucidate the text.

No physician, and especially those treating children, should fail to acquaint himself with the recent devices for preventing deformity after palsy, and no surgeon should undertake to treat these resulting deformities without a careful consideration of the newer methods described in this monograph. J. H. G.

A MANUAL OF THE PRACTICE OF MEDICINE. PREPARED ESPECIALLY FOR STUDENTS. By A. A. STEVENS, M.D. Philadelphia, New York and London: W. B. Saunders & Co., 1903.

THIS excellent manual is well known to several generations of students within and without the college walls. The many additions and alterations in the text of this (the sixth) edition, rendered necessary by the rapid progress of medicine in these days, are presented with all the author's usual clearness and conciseness of style, which he has acquired by years of practice in guiding the learner's footsteps along the difficult and crowded paths of medicine. Much of the text has been practically rewritten for the present edition, especially the section on diseases of the digestive system. Here the classification has been simplified and such special divisions as "dyspepsia," "peritonitis," and "ascites," very properly suppressed altogether. A little more attention might perhaps have been devoted in this portion to the physical examination of the abdominal organs, especially the stomach and colon. This subject is not formally treated, and we note with some regret that the methods of determining the size of the stomach by inflation have not been considered worthy of mention; the practice of distending the organ with carbon dioxide by administering the two parts of a Seidlitz powder separately is referred to as a routine procedure, it is true; but, as recent developments have shown that it is not always free from danger, some fatalities having been reported after its use, the subject is deserving of some comment. The article on malaria, as was to be expected, appears in a new garb, in keeping with the most recent fashion in etiology and pathology, although the failure to mention microscopic examination of the blood for diagnostic purposes must be characterized as an omission, even in the most compact manual on the practice of medicine. However, the imperious necessity of keeping the volume within certain limits, like charity, would cover many more sins of omission than could be found even by the most carping critic. R. M. G.

CLINICAL EXAMINATION OF THE URINE AND URINARY DIAGNOSIS. A CLINICAL GUIDE FOR THE USE OF PRACTITIONERS AND STUDENTS OF MEDICINE. By J. BERGEN OGDEN, M.D. Second edition, thoroughly revised. Philadelphia, New York and London: W. B. Saunders & Co.

THE special feature of this book, which distinguishes it from others of its type, is the thorough and systematic discussion of the clinical significance of urinary changes. The second portion, which embraces almost one-fourth of the text, is devoted to diagnosis, both of the different forms of kidney disease and of diseases outside of the kidneys, by means of urinalysis. The list of diseases is complete, embracing both general and local disturbances, whether medical or surgical; in short, this part of the work is a complete clinical guide to urinary diagnosis. We do not know any other work that treats this part of the subject so exhaustively. By simply referring to the table of contents the known facts about the urine in any given disease can be ascertained at once and without a tedious search through the entire list of individual normal and abnormal urinary constituents. A short abstract of the clinical symptoms is, of course, added. If the statements in this portion of the book are based on actual observations, either made by the author himself or collected from the literature, they present a vast amount of research and reading, which is, however, fully repaid by the value of the results. One is impressed, after reading the book, by the importance of continuous study of the urine throughout the course of a disease. The second edition has been brought up to date in every respect; even cryoscopy, one of the most recent developments in urinology, finds at least a passing mention. In the classification of diseases of the kidneys the author follows Councilman and the most recent pathologists, whose nomenclature he has adopted, giving the older terms as well, however, for the sake of identification. Thus, "chronic parenchymatous nephritis" has now become "subacute glomerular"; in fact, "parenchymatous" has disappeared altogether from the nomenclature and is replaced either by "diffuse" or by "glomerular." This classification, being based on pathological findings, cannot always be strictly followed in practice, however, as the author himself concedes; but it helps to clarify one's ideas of urinary pathology. That portion of the book which is strictly devoted to laboratory work contains nothing new of any importance; but several of the traditional tests have been discarded. The article on albumin shows careful selection and is particularly well arranged; the directions for detecting albumin include a paragraph on the approximate estimation of albumin in which the author distinguishes eight degrees in the reaction of albuminous urine to the nitric acid test, making it possible to dispense with quantitative estimation in practical work. The book is well illustrated with a

number of colored plates and numerous cuts, and contains much valuable information of a practical sort about the choice and management of apparatus and the best sources from which to obtain them, with many other useful hints. R. M. G.

A Reference Handbook of the Medical Sciences Embracing the Entire Range of Scientific and Practical Medicine and Allied Science by Various Writers. A new edition, completely revised and rewritten. Edited by Albert H. Buck, M.D. Volume VII. Illustrated with chromolithographs and 688 half-tone and wood engravings. New York: William Wood & Co.

In a review of one of the volumes of a work such as this, it is impossible to adequately consider all of the individual contributors. Suffice it to say that no article has come under the reviewer's notice, after a careful search throughout the book, that does not show the most careful work on the part of its author and a most judicious editorial supervision on the part of Dr. Buck. A due sense of proportion prevails throughout, and no subject is dismissed with too slight notice, nor is there undue expatiation upon what might be regarded as unimportant matters.

The present volume contains some contributions which may be singled out as authoritative monographs upon the topics considered in them. We would mention especially the contribution on Teratology by Piersol, probably the best known authority on this subject in the world. In addition to the valuable text, Dr. Piersol contributes a number of most interesting illustrations of monsters, and of the method of their embryological development. The article on Tuberculosis by Councilman is also an especially noteworthy and authoritative exposition of the subject. Dr. M. Allen Starr writes, as might be expected, a most lucid account of the description of the diagnosis of local tumors of the spinal cord. The articles upon the Spleen, by Mall; the Sympathetic Nervous System, by Onuf; and that upon Syphilis, by James Nevins Hyde, are all worthy of the high reputations of their authors.

It speaks well for the editorial ability of Dr. Buck that he should have been able to secure the services of such men as Pilcher to write upon Tracheotomy, and Roswell Park upon Trephining, or, as he apparently prefers to call it, Trepanning. D. Bryson Delavan contributes a most interesting description of the tonsils, and the treatment of their various pathological lesions.

The physician who is fortunate enough to own a set of the Reference Handbook may congratulate himself upon having

acquired a library which may be considered a *multum in parvo* of medical literature. Although the volumes are very bulky they are not unwieldy, and their clear print and handsome appearance places them far above most works of an encyclopædic nature.

F. R. P.

THE DECENNIAL PUBLICATIONS, UNIVERSITY OF CHICAGO. A Description of the Brains and Spinal Cords of Two Brothers Dead of Hereditary Ataxia. Cases XVIII. and XX. of the Series in the Family Described by Dr. Sanger Brown; with a Clinical Introduction by Dr. Sanger Brown. By LEWELLYS F. BARKER, M.D., Professor and Head of the Department of Anatomy. Printed from Volume X. Chicago: University of Chicago Press, 1903.

THE rare and valuable material used as a basis of study for the present monograph is described and worked up with the greatest care and precision. The text is excellently and very elaborately illustrated. Dr. Brown in the clinical introduction gives a résumé of the symptomatology of hereditary ataxia, to which is added a family tree of the cases described by him. A careful clinical report of the two fatal cases follows.

The main portion of the monograph is given up to a study made by Dr. Barker of the topography of the brain and microscopic lesions of the brains and cords of the two brothers. The brains and cords of both cases were relatively small, the cords, medulla, and pons in each case looking smaller in proportion than the cerebrum. The various sulci and gyri showed no deviation from the normal type which could be regarded as standing in relation to the disease from which the family suffered.

The microscopic study revealed degenerations of one system of exogenous fibres of the posterior funiculus (apparently corresponding to the third fetal system of Trepinski), the dorsal nucleus of Clark, the direct cerebellar tract of Flechsig in the lateral funiculus, and its continuation in the restiform body. In addition there was some involvement of the dentate nucleus of the cerebellum and the brachium conjunctivum, and probably also of the inferior olivary nucleus of the medulla oblongata. Corresponding to the degeneration of the white matter of the spinal cord there was an increase in the glia tissue. These lesions correspond almost exactly with what Dr. Adolf Meyer found in another instance from the family which he studied. The degenerations in the posterior funiculus should probably be regarded as systemic. Barker believes that they involve only certain of the stem fibres and collaterals of the intramedullary continnation of the posterior roots, namely, those corresponding to one period of medullation in the embryo.

W. T. L.

MAMMALIAN ANATOMY, WITH SPECIAL REFERENCE TO THE CAT. By ALVIN DAVISON, PH.D. Philadelphia: P. Blakiston's Son & Co., 1903.

THIS little work is an introduction to the study of mammalian anatomy, and as such is sure to prove of great value to teachers and students, especially those working in high schools or educational institutions of a similar nature. Although the book is small in size, it is very thorough and accurate, and the illustrations are particularly worthy of commendation. Each section is followed by a number of "practical questions and suggestions" which will prove very helpful in the classroom. The author writes in a style which is both lucid and concise, and, while popular, cannot be termed unscientific. J. H. G.

A HANDBOOK OF OPHTHALMIC SCIENCE AND PRACTICE. By HENRY E. JULER, F.R.C.S., Ophthalmic Surgeon to St. Mary's Hospital; Consulting Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Ophthalmic Surgeon to the London Lock Hospitals. With illustrations. Third edition, revised and enlarged. Philadelphia and New York: Lea Brothers & Co., 1904.

THIS work is a satisfactory exposition of modern ophthalmology so far as the subject is capable of presentation in a work of its size. A special feature is the free use of illustrations, both chromolithographs and wood-cuts; while the former are perhaps somewhat overcolored in some cases, the work gains the advantage of being almost an illustrated atlas of the eye.

The seventeen chapters of which it consists, besides appendices, begin with the eyelids and take up in order the diseases of the ocular structures and adnexa; 130 pages, about one-fifth of the whole, are devoted to refraction. Optical fundamentals and the different varieties of the refraction of the eye with a description of the function of accommodation are clearly stated without making special demands upon mathematical knowledge.

It is interesting to compare certain points of practice which are no doubt quite general in England with the corresponding practice in this country. Thus in myopia the author states: "A mydriatic is not as a rule necessary except in young children or when astigmatism is present. After the age of thirty years a mydriatic is seldom necessary." In ordering glasses for hypermetropia the author advises that 1 D. be deducted from the total hypermetropia under the age of twenty years, half a D. between twenty and thirty years and full correction after that age. If the hypermetropia be above

4 D. an additional half or 1 D. must be deducted according to the degree and age. The author endorses the view that myopes of whatever degree should wear glasses constantly as near the full correction as possible. He is somewhat non-committal upon the operation of high myopia and truly says that it is as yet impossible to state definitely whether the progress of the myopia is arrested by the operation.

Presbyopia is corrected by the rule of thumb method which gives so much per year. This method of correcting presbyopia is unfortunately not unknown in this country.

We are pleased to see that heterophoria is given the very subordinate place which it deserves: only five pages. Graduated tenotomies are not mentioned.

The appendix contains 43 formulæ, embracing caustics, collyria, unguents, etc.; 8 interesting plates, semidiagrammatic, by G. S. Keeling, illustrate the normal and pathological histology of the eye. A glossary of Greek derivatives (a few Latin) is an aid to the comprehension of ophthalmological terms. T. B. S.

BIOGRAPHIC CLINICS. Volume II. The Origin of the Ill Health of George Eliot, George Henry Lewes, Wagner, Parkman, Jane Welch Carlyle, Spencer, Whittier, Margaret Fuller Ossoli, and Nietzsche. By GEORGE M. GOULD, M.D. Philadelphia: P. Blakiston's Son & Co., 1904.

IN this volume the author continues the studies of the ill health of distinguished literary people, of which a number were presented in a previous volume. Those whom he now presents are George Eliot, George Henry Lewes, Wagner, Parkman, Jane Welch Carlyle, Spencer, Whittier, Margaret Fuller Ossoli, and Nietzsche. By a careful study of his subjects' life history, as detailed not only in their biographies or autobiographies, but in their works, Dr. Gould has come to the conclusion that in every instance the physical suffering and its induced morbidity, which was apparent in their lives and labors, depended upon eye-strain, which could have been corrected in every case by properly fitted glasses. When one considers the vast variety of symptoms which these various authors presented, it is rather a hard task to bring one's self to believe that every one of them originated from a single common cause. Dr. Gould, however, adduces many circumstances which prove that in most instances, at any rate, there was considerable trouble with the eyesight, and much relief might have been given by proper adjustment of glasses, though it must be confessed that it is rather hard to believe that all their complaints were solely due to eye-strain.

Dr. Gould is certainly a bold man. In his preface he criticises the critics of his previous volume, and candidly states that he thinks the members of the medical profession are not in possession of a true summary of the facts contained in his books because of the "malfunction of their journalists and reviewers." He also charges the neurologists and ophthalmologists with neglecting or treating with contemptuous silence what he considers that he has proved. He reproaches the medical profession in general for neglecting the study of morbid physiology or functional disease in the ardor with which they pursue organic and infectious diseases.

Although we cannot feel that Dr. Gould has proved his point in all the cases which he has stated, nevertheless he makes out a very strong case, and his book will be of value if it can only make the profession realize how much functional disorders of the eyes have to do with the general health.

The work is of interest also from a literary point of view, as a pathological study of certain well-known authors. F. R. P.

ELEMENTS OF SURGICAL DIAGNOSIS. By A. PEARCE GOULD, M.S. LOND., F.R.C.S. ENG., Surgeon to the Middlesex Hospital; Member of the Council of the Royal College of Surgeons of England, and of the Examining Board for England; Member of the Senate of the University of London. Third edition, revised and enlarged. Chicago: W. T. Keener & Co., 1903.

THIS condensed work, presenting the Elements of Surgical Diagnosis, is well known through its previous editions. The present edition shows much revision and much new material, which adds greatly to its value. Although the book is small, there are no illustrations and the print is not large, therefore a vast amount of material is found between its covers. Surgical diagnosis is a subject which needs attention from all practitioners, and it will be found here presented in a very terse and comprehensive manner. Among the changes noted in this edition is a section on intracranial complications of middle-ear disease, which has been added to the chapter on Diseases of the Head. Notable changes are also found in the portions devoted to the abdomen. The present edition will find the same place in the esteem of both the student and the practitioner which its predecessors have won. J. H. G.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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The Cardiac Dulness in Cases of Cancer.—W. GORDON finds that in cases of cancer, particularly where the alimentary canal is involved, there is a remarkable diminution and often a complete obliteration of the area of cardiac dulness when the patient is in the recumbent posture. The possibility of emphysema was always excluded. The writer has observed it in a large number of cases, but gives notes of only 6 to illustrate the sign. He became so convinced of the value of the sign that he risked the diagnosis of cancer on the strength of it, where, otherwise, the evidence was inconclusive, and in 5 consecutive cases the diagnosis proved correct. In non-cancerous cases without emphysema he has failed to find it with three exceptions. It does not occur in sarcoma. Gordon states that all one can claim for it is that when present in a doubtful case it is highly suggestive of the presence of cancer, and when absent at a late point in the case its absence is somewhat suggestive of the absence of cancer.

The writer at first thought that the diminished cardiac dulness was due to diminution in the size of the heart commensurate with the general wasting, but the autopsy findings did not bear this view out. He is inclined to believe that the true explanation is a lessening of the elasticity of the lung tissue, comparable to the loss of elasticity of the skin of the abdomen in cases of visceral carcinoma. This loss of lung elasticity causes a sort of false emphysema, in consequence of which the edges of the lungs gradually close over the front of the heart.—*Proceedings of the Royal Medical and Chirurgical Society, March* 5, 1904.

On the Albuminuria of Puberty.—LOMMEL (*Deutsch. Arch. f. klin. Med.*, Band lxxviii., Heft 5 and 6, p. 541) contributes some interesting observations upon the urine of 587 young employés in the factories of Zeiss and Schott in Jena. The patients were between the fourteenth and eighteenth years. The majority of these patients were under observation from three to four years, the ages being from fourteen to eighteen

years. 111, or 18.9 per cent., showed either once or repeatedly a positive reaction for albumin. In the great majority of cases the albuminuria was intermittent, appearing often at periods far removed from one another. Examinations were made in most cases by acetic acid and ferrocyanide of potassium, part of the earlier examinations having been made by boiling. The amount of albumin was generally very small, and consisted in most cases apparently of serum globulin as well as serum albumin. The amount estimated by Esbach's method was usually from 1.20 to 1.10 per cent., rarely over 1.10 per cent. As a rule, there was no appreciable sediment; sometimes hyaline casts and occasionally fatty epithelial cells were found. Lommel considers the cause of the albuminuria of puberty as in part an evil condition of the blood, in part a slight degree of cardiac insufficiency, with a tendency to passive congestion, diseased circumstances which depend upon puberty in that at the time of the greatest growth of the body and the most active period of development, the new formation of blood does not keep proper pace with the other changes, nor does the heart become fully adapted to the increased demands dependent upon the rapid growth of the body and the correspondingly vigorous development and increase of energy. Lommel was struck by the fact of the very common evidence of cardiac and vascular disturbances among his cases, a part showing the juvenile growth of hypertrophy of the heart described by Sehl and Krehl. Bodily exertion seemed to have little effect on the albuminuria, the early morning urine showing essentially the same features as that of the evening. The early morning examinations were, on the whole, less apt to reveal albumin than those later in the day.

In only two instances he was able to examine into the question as to whether the albuminuria was or was not orthostatic. In each of these instances rest in a horizontal position resulted in the disappearance of albumin, which returned in the erect posture. He was unable to extend his examinations beyond the eighteenth year, but out of 130 patients there were over 25 belonging to just the same class as those examined by Lommel. Professor Matthes found albuminuria in but one instance.

How can the functional albuminuria be distinguished from that dependent upon true organic changes? This Lommel considers extremely difficult, and it can be done only through the possibility of long observation and careful history. It may be asserted that physiological albuminuria is rarely present to the extent above 1.20 per cent., while the presence of epithelial and granular casts speaks in favor of an organic disease. On the other hand, a clear cycle in the excretion of albumin is in favor of the functional character of albuminuria. Of especial interest, however, is the following statement of the author: "On the other hand, from my observations left-sided heart hypertrophy and abnormal tension of the pulse do not possess the importance from a point of view of differential diagnosis, which is frequently ascribed to them as indications of an interstitial nephritis."

In the great majority of his 111 cases further observations showed a great improvement in all the symptoms, including those relating to the heart and vessels.

Observations upon Koplik's Spots, Diazo-reaction, and Fever in Measles.—MULLER (*Münchener med. Wochenschrift*, 1904, No. 3, 51st Jahrg) had an opportunity to study an epidemic of measles involving

25 cases, which occurred in Marburg in the winter of 1902 and 1903. The epidemic spread very rapidly, as is shown by his curve, the cases increasing from 4 on the first three days to 18 on the fourth. The character of the epidemic was mild; complications occurred in 24 per cent. of cases. The mortality was 2.8 per cent. Six children died of bronchopneumonia. Most of the patients were four years old or under, the elder children having acquired immunity during a previous epidemic. In 166 of the Marburg cases Koplik's spots occurred 134 times, that is, in 81 per cent. An initial exanthem on the hard palate was observed 143 times, that is, in 86 per cent. of the cases. Since the great majority of all the patients were first brought to the polyclinic about the time that the exanthem began, it was impossible to say whether the Koplik spots preceded the appearance of the exanthem. In only 12 cases was it possible to follow the children with reference to this point from the incubation period to the outbreak of the disease. Of these 12 cases, Koplik's spots appeared 7 times simultaneously with the first rise of fever, that is, on the first day of the disease, 4 times one or two days after the occurrence of fever, and once the spots were absent altogether. In these 11 cases the Koplik spots appeared about 8 times from a half to two days previous to the initial exanthem, and 3 times the spots and exanthem occurred simultaneously.

The epidemic of measles, which ended in April, was followed in June and July by a little epidemic of rötheln. The diagnosis of rötheln was all the easier to make, as the greater number of the cases affected the same children who had recently recovered from measles. Fourteen cases of rötheln came under observation. In 6 of these Koplik's spots were definitely demonstrable. Nothing could be learned as to the time of the appearance of these spots, as all the cases were first seen with the full-blown exanthem. If one considers measles and rötheln as different diseases, it is evident that these cases prove that Koplik's spots alone are not diagnostic of measles.

A series of 10 cases were studied with regard to the diazo-reaction. Of these 10 cases, 8 showed the reaction first with the appearance of the exanthem, 2 half a day sooner. The intensity of the reaction increased with the duration of the continued fever and reached its highest point either with or soon after the maximum temperature. The reaction disappeared during the first days, at the latest six days after the disappearance of fever.

The fever curve of measles was also observed. In 12 cases where brothers and sisters of sick children contracted the disease the curve was followed from before the onset. Of these 12 cases, 9 showed the fever curve that is ordinarily given as a typical one in text-books. This type starts with a sudden rise. This is followed by a one to two-days' intermission, and then by a continued fever for several days. In 2 cases a more gradual remitting type of onset was seen, such as Heubner has described. In only 1 case did the fever rise suddenly without any previous remission.

Müller concludes his article, which gives an extensive review of the literature, as follows: 1. Koplik's signs are present in four-fifths of the cases of measles, and are frequently present on the first day of the disease. They are not, however, diagnostic of measles alone, as they were repeatedly observed in rötheln. 2. The diazo-reaction in the urine can be demonstrated almost always at the height of the disease. It occurs

usually with the appearance of the exanthem, and is not an early symptom. 3. In most of the uncomplicated cases of measles the fever curve shows a sharp rise at the beginning of the catarrhal stage. This rise is followed by an intermission, lasting one or two days, which, in turn, is succeeded by a rapid rise and continuous fever, lasting about four days, which falls usually by crisis. It is more seldom for the fever to rise by gradual remission or for it to start with a sudden unchecked rise.

SURGERY.

UNDER THE CHARGE OF

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Camphoric Acid as a Prophylactic for Urinary Fever.—FREUDENBERG (*Septième session de l'association francaise de urologie*, Paris, 1903) states that when he first practised the Bottini operation he frequently had cases that developed urinary fever—a complication alarming to the patient and disagreeable to the surgeon, as it varies in severity from a simple and relatively benign urinary fever to a true pyæmia in which the prognosis of necessity became extremely grave. Clinical experience has shown that the internal administration of quinine, salol, urotropin, methylene blue, etc., is absolutely without benefit, but that camphoric acid in three-grain doses, beginning some days before and continuing for some days after the operation, is an admirable prophylactic.

Intravesical Injections of Iodoform in the Treatment of Ammoniacal Cystitis.—FREUDENBERG (*Septième session de l'Association française de urologie*, Paris, 1903) states that while nitrate of silver and other agents have their principal action upon the bladder wall, the iodoform acts almost entirely in modifying the alkalinity of the urine, and, therefore, its use is not indicated in those cases of cystitis with acid urine nor in vesical tuberculosis. Its action is rapid. After one or more injections the masses of ropy pus and the ammoniacal odor disappear, the pain becomes less severe, and the desire to urinate frequently less urgent, while a cystoscopic examination will show that the mucous membrane is much less hyperæmic than before the injections were begun. The dose should be 0.3, suspended in glycerin, and then well diluted with some bland fluid, such as boric acid solution, and injected about every other day; but care should be taken to closely watch the patient, and at the first sign of intoxication the treatment should be stopped.

Wounds of the Stomach by Fire-arms.—FORGUE and JEANBRAU (*Revue de chir.*, 1903, No. 12), after an admirable and most complete review of the entire literature of this subject, state that every case demands immediate operative interference for its relief. This should consist in a median supraumbilical laparotomy, followed by an exploration of the anterior surface of the stomach, the liver, the neighboring coils of intestine, the spleen, all the accessible portion of the diaphragm, and the posterior surface of the stomach. All visceral wounds should then be closed by suture, the toilette of the peritoneum carefully completed, and then the abdominal wound closed after proper provision for drainage has been made.

Osteomyelitis of the Coccyx.—MONNIER (*Rev. de orthopédie*, 1904, No. 2) refers first to the interesting article by Grisel in the November issue of this JOURNAL on the "Primary Acute Osteomyelitis of the Vertebræ," in which he refers in detail to the forty-three cases of this disease which presented themselves for treatment at Kirmisson's clinic. In this series is included a note of six cases in which the sacrum was involved, and the seriousness of the disease may well be appreciated, for of these cases not one made a recovery. Monnier notes in detail one case in which the coccyx was involved, but which made a good recovery after operative interference.

Penetrating Wounds of the Heart; Suture; Death.—VINCE (*Jour. de chir. et annales de la soc. belge de chir.*, 1903, No. 7) states that the external wound was in the fifth left intercostal space, in the parasternal line, horizontal in its direction, and 2.5 c.m. long. Its borders were clean-cut, no bleeding externally nor bloody expectoration, no subcutaneous emphysema; the edges of the wound were closely approximated; the left side of the chest moved but little during respiration. On proceeding to the examination of the chest there was found dulness upward to the inferior angle of the scapula with all of the physical signs of a vast pleural effusion. An operation being decided upon, an incision was made with its superior border corresponding to the wound, the internal border corresponding to the left edge of the sternum and the inferior border corresponding to the superior edge of the eighth rib. This hinge-like skin-flap with the underlying cellular tissue was dissected backward. Then in the seventh interspace was found a gaping wound from which air and blood escaped with the respiratory movements. A musculo-osseous flap was then made, and the fifth, sixth, and seventh costal cartilages were cut. After turning them back the pleura was freely incised and found filled with blood, partially clotted. At each expiration the pericardium appeared in the wound. The lung was found retracted toward the vertebræ upon its hilum. The pleura was rapidly cleansed, and then the 2-c.m.-long wound in the pericardium was plainly visible. Upon separating the edges of the pericardial wound, it was found that there was a wound near the apex of the left ventricle, from which the blood "foamed" at each diastole. This wound in the ventricle was closed, using an intestinal needle and No. 2 silk, with three interrupted sutures, one of which perforated a coronary branch, necessitating its ligation. All hemorrhage being arrested, the pericardium was washed out and then closed with catgut sutures without drainage. The wound was then closed, and

the patient returned to bed, being much shocked. Salt solution and stimulants were freely used, but without avail, death following two days later.

Some points in this case would seem to merit further attention. 1. In regard to the difficulty of making a diagnosis. The most of the physical signs and symptoms presented by this case were simply of the presence of an intrathoracic hemorrhage, but the organ that was injured could not with certainty be discovered without operative interference. The only possible diagnosis was one of rapidly increasing hæmothorax, which demanded an immediate operation for its relief. 2. The patient bore chloroform anæsthesia very well. 3. The horse-shoe-shaped flap with the temporary resection of the costal cartilages proved valuable in that it widely exposed the field of operation. 4. In all cases of suture of the pericardium one should free the mediastinal portion of the pleura that is in contact with the pericardium, so that a separate suture of these two serous membranes can be made, which tends to limit the chances of their being eventually infected. 5. The pericardium was not drained, and it would seem to be best not to do so unless there existed some especial indication, for drainage is an essential factor in the production of pericardial, adhesion, and it does not prevent infection. The greater number of the fatal cases are those in which drainage was employed. However, drainage of the pleura is usually indicated by reason of the great ease with which this membrane becomes infected. This marked liability to infection is without doubt the greatest danger in all cases of cardiac wound.

THERAPEUTICS.

UNDER THE CHARGE OF

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Adrenalin in Gastrointestinal Hemorrhages.—Dr. M. H. Schlesinger has successfully treated two patients suffering from hemorrhage of the alimentary tract by adrenalin given internally. The first patient was a hæmophile, with severe intestinal hemorrhage. Six minims of a 1 : 1000 solution, given every hour, caused a cessation of the bleeding. In twenty-four hours the patient took 7½ drachms of the solution. After further administration of the agent, in connection with gelatin, a drop of blood obtained by puncture coagulated at once. The second patient suffered from essential purpura, with bleeding from various mucous membranes, especially that of the stomach. This was stopped by the administration of adrenalin given as in the first case. The author has employed the drug in various other forms of alimentary hemorrhage, with invariable success. Hæmoptysis, on the other hand, is not influenced by this treatment. Internal administration of adrenalin has no effect upon blood pressure.—*La semaine médicale*, 1904, No. 11, p. 86.

Tincture of Horse-chestnut in Hemorrhoids.—Dr. de Veney recommends the tincture of the horse-chestnut in all forms of this affection. Its first effect is to lessen the pain, which it does after a few doses, but if permanent results are to be accomplished the remedy must be continued for some time after the cessation of the pain. The dosage should be at least 10 drops in a little water before the two principal meals of the day, but the dose should be regulated according to the intensity of the symptoms. When these are severe and accompanied by marked turgescence of the tumors, 20 to 30 drops may be given, which quantity should be gradually lessened as the pain ameliorates. In cases with mucoid and dysenteric stools, blood, and tenesmus, the drug should be given with equal parts of tincture of aloes or in connection with a pill of $\frac{1}{10}$ of a grain of silver nitrate taken night and morning. For patients with intestinal atony and constipation equal parts of tincture of nux vomica should be given as an adjunct. The medicament may also be applied locally in an ointment, but its effects are not so marked when used thus as when given internally. Tincture of horse-chestnut is also useful in varices of the legs and in congestive disorders of the pelvis, such as inflammation of the neck of the bladder, prostatitis, proctitis, and uterine congestion.—*Journal de médecine de Paris*, 1904, No. 13, p. 126.

Electricity in Habitual Constipation and Mucomembranous Colitis.—M. Delherm has used in constipation a galvano-faradic current of over 50 milliampères with good results. In 46 out of 53 patients he has brought about regular spontaneous defecations and has modified the constipation, both quantitatively and qualitatively. Of 16 patients whose stools contained mucus this morbid condition disappeared in 13 and was ameliorated in 3. In all cases an increase of weight was noted. The results obtained lasted permanently in 36 of the cases. Cases of mucomembranous colitis were usually benefited by the treatment, the constipation disappearing and the mucus becoming less in quantity. M. Zimmern employs in membranous colitis a constantly varying continuous current with a long sinusoidal curve. With this he obtains satisfactory results, both immediate and remote. The membrane, the pain, and the constipation disappear in order. Dieting has no part in the treatment. Laxatives are given (castor oil or an enema), if necessary, about once in five days until the stools appear spontaneously. A regular hour for defecation is insisted upon, and at this time a small enema of cold water may be given, so as to excite the rectal reflex. The author does not consider that the electric current excites the intestinal musculature, but that it acts upon the vascular supply of the bowel through the solar system.—*La presse médicale*, 1904, No. 27, p. 215.

Sulphanilic Acid in Catarrhs of Mucous Membranes—M. Vautri states that this agent relieves acute laryngitis and causes the pain of otitis media to disappear rapidly. The neuralgic pains accompanying inflammations of the various mucous membranes rapidly disappear under its administration, but in true neuralgias the drug is useless. In chronic conditions it may be given for considerable lengths of time, but in these it is less effective than in acute inflammations. When taken in doses of from 15 to 20 grains per day for several weeks, it causes no digestive or other disturbance, except at times a slight diarrhœa. In

acute coryza a dose of ½ to 1 drachm is effective within two hours: The following is a good formula for the administration of the drug: Pure sulphanilic acid, 10 parts; sodium carbonate, 8½ parts; distilled water, 200 parts: from 5 to 10 drachms daily, preferably in two doses.—*Journal de médecine de Paris*, 1904, No. 11, p. 103.

A New Treatment for Asthma.—DR. SAENGER has reported good results from the treatment of asthma by means of respiratory exercises. He bases his theory upon the facts that the instinctively deep respirations during an asthmatic attack augment the dyspnœa. During the attack the lung is the seat of an acute emphysema and of an alveolar distention which result in a diminution of the movements of the thorax. The deep inspirations made by the patient increase the quantity of residual air, and this also increases the immobility of the chest wall and exaggerates the dyspnœa. To combat this the patient ought to inspire as little air as possible and to make his expirations as long and as slow as he is able. Few and shallow inspirations and long and effortless expirations are to be attempted. The patient is advised thus, and is told that there is no danger of asphyxia. The slow and prolonged expiration is difficult, and the patient is told to whistle for three or four seconds before taking a shallow inspiration, or to count in a loud voice up to five and then inspire and to continue this scheme for ten or fifteen minutes. Certain cases are able to count as high as ten before inspiring. Another advantage of the method is that it takes the patient's mind from his dyspnœa. Between attacks it is advisable for the patient to practise the procedure daily for fifteen minutes. With practise it becomes possible to count even to forty before taking an inspiration. The author has employed the treatment in 11 cases, in 9 of which it has achieved remarkable results; the habitual respiratory oppression was greatly lessened or disappeared entirely, the intervals between attacks were lengthened, and at the same time, the bronchitis and emphysema were favorably influenced.—*Münchener medicinische Wochenschrift*, 1904, No. 8, p. 335.

The Treatment of Alopecia Areata.—M. CHARMEIL recommends the following treatment: In marked cases the entire scalp should be clipped. Pure acetic acid should be applied to the bare areas by means of a small cotton swab. This should be quickly applied and the seat of the application at once dried with cotton. The skin immediately becomes blanched, but within a few days an intense redness appears and sometimes a small blister; after several days the skin resumes its normal appearance. The patient should be given a prescription, which is to be applied with friction every two or three days, such as follows: Ether, 6 drachms; chloral hydrate, 15 to 60 grains; crystalline acetic acid, 8 to 30 grains. This maintains the counterirritation of the skin. Other counterirritants, such as chrysarobin, tincture of iodine, croton oil, or solutions of carbolic acid, may be employed. In rebellious cases some vesicant in the form of a plaster may be applied, and the raw surface touched with silver nitrate solution, 1 : 15. Care should be exercised not to blister so deeply as to cause cicatrices. The whole scalp should be shampooed twice a week and a solution analogous to the following rubbed in: Alcohol, 90 per cent., 300; tincture of lavender, 100; tincture of melissa, 100; mercury bichloride, 0.006; tincture of cantharides,

15; chloral hydrate, 5; castor oil, 2. In addition, the employment of massage, vigorous brushing, and faradization are useful.—*Journal de médecine de Paris*, 1904, No. 14, p. 138.

Arsenous Acid in Sleeping Sickness.—M. A. LAVERAN states that arsenous acid, which has given favorable results in the treatment of this disease, will cause the trypanosoma to disappear temporarily, at least, from the blood of infected rats. The proper dose is one part for each 20,000 of the animal's weight. Smaller dosage than this produces an incomplete effect or none at all. A solution containing 1 gram of arsenous acid and the same quantity of sodium carbonate to 500 grams of distilled water is used. Possibly this treatment might be applicable to human beings infected with the parasite in the phase of the affection which precedes the appearance of nervous disturbances. It is better to give large doses at intervals than smaller daily doses, as has hitherto been the custom.—*La semaine médicale*, 1904, No. 9, p. 68.

Treatment of Neuralgia.—DR. BASTIE reports remarkable results from the following treatment. He uses extract of valerian, 3; hyoscyamine, 0.045, or extract of hyoscyamus, 0.75; morphine hydrochlorate, 0.15. These formulæ are made up in pill form, and one pill is given at nine or ten o'clock in the evening and a second a half hour later. A third is given at five in the morning and a fourth at eight. These are sufficient to relieve the pain completely. If there is a recurrence the patient may take a pill each morning and evening for eight days. All neuralgias, when uncomplicated, yield to this treatment. Between the attacks 15 grains of quinine are administered twice a day. When there is congestion of the involved nerve the treatment is supplemented by leeches applied at the point of emergence.—*Revue française de médecine et de chirurgie*, 1904, No. 7, p. 161.

OBSTETRICS.

UNDER THE CHARGE OF

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Induced Labor, with Especial Reference to the Permanent Results for the Children.—LOREY (*Archiv für Gynäkologie*, Band lxxi., Heft 2, 1904) contributes an interesting and instructive paper upon this subject. The most frequent indication for the induction of labor is contraction of the pelvis, and Lorey reports the results of 100 cases.

The most frequent variety of contracted pelvis was the flat rachitic; next in frequency, the justo-minor, and after that the irregularly contracted pelvis. Of these 100 children, 74 were born living, or 74 per

cent. Of these 19 per cent. died during the first ten days of life; 60 per cent. left the hospital living. The subsequent history of 56 children was obtained after they left the hospital, and 71.5 per cent. of these children survived the first year. Of 51 illegitimate children, 78.5 per cent. lived through the first year.

These operations were performed upon 83 women, 88 per cent. of whom recovered without fever. One patient died of septic infection; the mortality of the mothers was 1.2 per cent.

Observation shows that the induction of labor in contracted pelvis is most successful in the interests of mother and child if it be not too long postponed and if the pelvis is but slightly contracted. In the interests of the child it should not be induced before the thirty-fifth week of gestation, and it must be considered as fatal to the child before this time as is craniotomy.

Pregnancy was also interrupted in thirty-seven cases because of some disease of the mother. Fourteen of these cases had eclampsia, 6 nephritis, 3 disease of the heart, 6 placenta prævia, 4 obstinate dyspnœa, 1 goitre, 2 polyhydramnios, 1 carcinoma, and 1 infection of the bladder produced by pressure of the head upon the urethra.

The mortality among these children was 36.8 per cent., while 63 per cent. left the hospital living. So far as could be traced, 70 per cent. of these children died during the first year of life, while but 30 per cent. survived the first year. It was estimated that in all but 15.8 per cent. of the children remained alive at the end of the first year.

Lorey would not advise the induction of labor in contracted pelvis where the internal anteroposterior diameter was 7 cm. or less. Where the internal anteroposterior diameter varied from 8 to 10 cm. induced labor is indicated when the patient's history shows a marked disproportion between the head and the pelvis. In these cases the operation should be done before the thirty-sixth week. The use of bougies or elastic dilators is advised. In cases where much interference is practised, the prophylactic injection of antistreptococcic serum is recommended. In all serious diseases affecting the mother, labor should not be induced in the interests of the child.

Nephrotomy in Eclamptic Anuria.—In the *Zentralblatt für Gynäkologie*, No. 15, 1904, SIPPEL reports the case of a multipara, aged thirty-five years, who had eclampsia and, after the delivery of the child, anuria, which did not yield to treatment.

Upon autopsy, a condition of both kidneys was found resembling glaucoma. The case seems one in which incision of the kidney or decapsulation might have given good results.

Labor Complicated by Fracture of the Pelvis.—In the *Archiv für Gynäkologie*, Band lxxi., Heft 2, 1904, BOSSE describes the case of a patient who had once tried to commit suicide by throwing herself from a window upon a pavement. She had fallen upon the left femur and ischium. The patient was pregnant at the time, had incomplete abortion, which was terminated by curetting. She had incontinence of urine and feces, with atrophy of the lower extremities, and after four months' treatment was able to leave the hospital, a positive diagnosis of fracture of the pelvis never having been made. The patient made a gradual recovery and was able to sit and walk without pain.

She was afterward admitted pregnant and at full term, and, as delivery failed, hysterotomy was performed. She made a good recovery.

On examination she was found to be markedly rachitic, with an external conjugate of 18.5 cm.; the left tuberosity of the ischium was 1 cm. higher than the right. The diagonal measurements of the pelvis were unequal, the conjugate diameter being 8 cm. and the true conjugate 5.5 cm.

A radiogram of the pelvis was obtained, when the right half was found normal. The left half of the pelvis seemed to be higher than the right, and the pelvic girdle was broken by three fractures. One had extended through the left sacroiliac synchondrosis, one horizontally through the pelvis, and a third between the ischium and pubes. An exostosis had developed, and the sacrum seemed to be much shortened. There had also been injury to the last three lumbar vertebræ. The case would be considered by some writers upon the subject as a double vertical fracture.

Treatment of Retroversion of the Gravid Uterus.—HERMAN (*British Medical Journal*, April 16, 1904) reviews the pathology of this condition, and quotes the statistics from the London Hospital and from St. Bartholomew's Hospital. In the former institution, from 1885 to 1893, there were 79 cases of retroversion of the pregnant uterus. In 74 cases, 40 showed spontaneous ascent of the womb when the patient was kept recumbent and the bladder emptied. In 33 patients the uterus was manually replaced. Two cases aborted; in both the uterus had been replaced. Two died from toxæmia. In 1 case a water-bag was used to replace the uterus, and it is questionable whether it produced an effect.

At St. Bartholomew's Hospital, from 1881 to 1892, there were 42 cases, in 25 of which the bladder was emptied and the uterus easily replaced. In 2 cases attempts at reposition failed, but the bladder was frequently emptied, and the uterus afterward assumed a normal position spontaneously. In 2 cases abortion was induced, and 3 cases aborted spontaneously. Two died from the effects of retention of urine on the bladder and kidneys.

In these two hospitals there were 115 cases, with 5 abortions, or 1 in 23. This is about one-fourth of the usual frequency of abortion.

Herman believes that practically the only danger from retroversion of the gravid uterus results from the retention of urine. He considers the one necessary treatment the frequent use of the catheter.

He has never seen a case of retroversion in the gravid uterus where the womb could not be pushed up. He believes that the finger carried into the rectum is most efficient in bringing about this result. He sees no occasion under any circumstances to resort to abdominal section.

Death from Pulmonary Clot following Operation for Ectopic Gestation.—HIND (*British Medical Journal*, April 16, 1904) reports the case of a multipara who was operated upon while in a desperate condition for ruptured ectopic gestation. There had been free hemorrhage with the discharge of much blood into the abdomen. Eleven days after the operation the patient seemed quite well, except that the breathing was more rapid than normal and the pulse somewhat feeble. Without apparent cause she suddenly collapsed and died.

At autopsy the wound was healed, there was no sign of septic infection, the peritoneum was healthy, and the stump of Fallopian tube was firmly closed by the silk ligature. The pulmonary artery contained a large clot, laminated on section, and hollow in parts.

GYNECOLOGY.

UNDER THE CHARGE OF

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Treatment of Cervical Metritis.—RICHELOT (*La gynécologie*, 1904, No. 2), reviewing the result of his treatment of glandular endocervicitis with caustics, admits that the results as regards subsequent cicatrization have not been satisfactory, although he claims that after the lapse of six months or a year the cervix will be restored to a normal condition.

Epithelioma Developing from the Stump of a Polypus.—CROISIER (*La gynécologie*, 1904, No. 2) reports a case of intrauterine polypus, the stump of which was situated so high up in the uterine cavity that he could not excise it per vaginam. Fragments of the growth removed for examination were pronounced by the pathologist to be epitheliomatous, so that he decided to extirpate the uterus. As the cervix appeared to be healthy, supravaginal amputation was performed instead, the patient making a good recovery.

Examination of the specimen showed that the neoplasm was a fibromyoma, the stump of which showed well-marked cancerous infiltration involving the subjacent muscular tissue. The writer properly questions the wisdom of leaving the cervix, under the circumstances.

Referring to the supposed infrequency of cancer and fibromyoma in the same uterus, he reports four cases in which the two conditions were associated. In his opinion a fibroid uterus forms a favorable soil for the development of malignant disease, probably due to the endometritis which is generally present.

Radical Cure of Cancer of the Uterus.—BAROZZI (*La gynécologie*, 1904, No. 2), after analyzing the statistics of abdominal and vaginal extirpation of the cancerous uterus, asks how long a time should elapse before a patient is pronounced cured. He concludes, with Winter, that five years should be regarded as the limit, though recurrence has been noted as late as seven or eight years after operation.

Myomectomy versus Hysterectomy.—BATIGNE (*La gynécologie*, 1904, No. 2) concludes a paper on this subject, with the statement that the conservative operation should be confined to cases of single tumors in

young subjects, especially when the patient has expressed a strong desire to have the uterus preserved in the hope of future pregnancy; also in cases in which the presence of the neoplasm in a pregnant uterus is a menace to the continuance of pregnancy and the subsequent delivery.

Cysts of the Clitoris.—CAZIN (*La gynécologie*, 1904, No. 2) reports a case in which he removed a large pedunculated tumor which grew from the prepuce of the clitoris. It was polycystic, the cysts being lined with prismatic epithelium and containing a viscous fluid. In the discussion of this paper before the French Surgical Congress similar cases were reported by Témoin and Tédenat.

Cancerous Degeneration of a Uterine Fibroid.—FRANCHOMME (*Soc. des sciences méd. de Lille*) reports the case of a patient, aged sixty-four years, with metrorrhagia, who several years before had had a uterine polypus removed. On entrance to the clinic a large bleeding neoplasm presented at the os externum. The diagnosis of epithelioma was made and the growth was removed. Microscopically it was found to be composed of fibromuscular tissue filled with masses of pavement epithelium.

The writer comments on Richelot's advice always to perform total hysterectomy, in order to avoid possible cancerous degeneration of the cervix.

Congenital Anomaly of the Fallopian Tubes.—RONSSE (*Annales de la soc. de méd. de Gand*) reports the case of a woman, aged twenty-six years, who had menstruated regularly since the age of sixteen, though the periods were scanty and painful. She had been married six years without conceiving. The uterus was normal, the os being small. Divulsion and curettement were performed without the desired results. A year later the abdomen was opened on account of persistent pain and evidence of ovarian disease. Both ovaries were removed for cystic degeneration. The tubes were normal as far as their distal ends, where they terminated in a cul-de-sac, both fibrinated ends being wanting. There was no evidence of inflammation in or around the tubes.

The writer regards this anomaly as a proof of the dual origin of Müller's ducts, the proximal part of the tube formed by the invagination of the peritoneal epithelium being absent, while the distal portion developing from the Wolffian canal was normal.

Vaporization of the Uterus.—HAUTKE (*Zeitschrift für Geb. u. Gyn.*, Band xlix., Heft 2), in a recent discussion of this subject before the Berlin Obstetrical Society, stated that this method of treatment was not free from danger, since he had known of two fatal cases. The indications, he believes, are limited, being confined to those cases in which all other methods of treatment had failed, especially untractable climacteric hemorrhages. The writer always satisfies himself that he has not to deal with an atrophic or thin-walled uterus before introducing steam into it, and is sure that ample dilatation of the cervix is maintained. Submucous fibromyomata form a contraindication, and he never employs this method of treatment in young subjects.

Mackenrodt, in continuing the discussion, held that vaporization was a distinct step backward in gynecological therapeutics, to be included in the same class with cauterization with chloride of zinc. He had been obliged to extirpate several uteri for hæmatometra due to cicatricial contraction of the cervix following such treatment. Climacteric hemorrhages, if not due to malignant disease, are referable to arterial sclerosis. How this condition could be cured by destruction of the endometrium he could not understand. Total extirpation of the uterus is not only safe, but more scientific in cases of recurrent bleeding. With submucous fibroids repeated curettement is better than vaporization, while if the hemorrhage is due to adnexal disease the latter treatment is certainly useless.

The speaker did not find that the great expectations which had been held in regard to the intrauterine use of steam had been realized, but in isolated cases of hæmophilia he acknowledged that it might be indicated, though he had never met with one himself.

Czempin said that he had used steam at a temperature of 100° to 101° C. for three minutes with marked benefit, and had never seen stenosis follow. He had always made it a practice, however, to pass sounds for six weeks afterward, in order to insure patency of the cervical canal. He disagreed with Mackenrodt with regard to arteriosclerosis as a cause of climacteric hemorrhages, and in any case the effect of vaporization was to contract bloodvessels and to cause shrinkage of the uterine parenchyma, as well as the endometrium.

Appendicitis Complicated by Ectopic Gestation.—Temoin (*La gynécologie*, 1904, No. 2) reports two cases of acute appendicitis associated with extrauterine pregnancy. In both the latter condition was not suspected, as the symptoms pointed clearly to perforation of the appendix. In both it was necessary to resort to median laparotomy, as the ordinary lateral incision did not give sufficient room. The diseased appendix and ectopic sac were fused together. One operation was performed on the fourth and the other on the fifth day after the initial attack, vaginal drainage being employed. Both patients recovered.

Endothelioma of the Cervix Uteri—Kirchgessner (*Zeitschrift für Geb. u. Gyn.*, Band xlix., Heft 2) concludes an article on this subject as follows: Contrary to the opinion of Gebhard, young subjects are more apt to be attacked with this disease than old. Endothelioma may develop in nulliparæ as well as in multiparæ. The clinical symptoms are the same as those of cancer, *i. e.*, hemorrhage and foul discharge. The neoplasm is usually nodular, of variable consistence. No case of recurrence after removal has been reported, in spite of Burst's statement that endothelioma is known to recur locally. It grows slowly and has a limited power of metastasis. A radical operation should be performed as soon as the diagnosis is confirmed microscopically.

Tubercular Infection of Ovarian Cysts.—Prusmann (*Archiv. für Gynäkologie*, Band lxviii., Heft 3) affirms that no genuine case of primary tuberculosis of the ovary has yet been described. In the case reported by him an ovarian cyst containing tubercles was removed, without any trace of the disease elsewhere in the pelvis or abdomen. In this instance infection may have occurred through either the genital tract or the lymphatics, *i. e.*, by coitus. The finding of giant cells in

the lymphatics of the broad ligaments led to a belief that these latter were the channels of infection. Cornet and Jani have shown what a prominent role coitus plays in the transmission of the disease, since the tubercle bacilli in the semen are especially virulent.

Fibromyomatous Lipoma of the Uterus.—SEYDEL (*Zeitschrift für Geb. u. Gyn.*, Band l., Heft 2) describes a uterus which was extirpated for malignant adenoma of the vagina. A small intramural growth was found at the fundus, which presented the appearance of an ordinary fibromyoma, but on section was found to be filled with masses of fat. Only ten other cases of uterine lipoma have been reported.

The origin of the fat has never been satisfactorily explained. The writer leans to the theory of inclusion of lipoblasts in embryonic life, thus agreeing with Merkel and Knox.

OTOLOGY.

UNDER THE CHARGE OF

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Precautions against Injury from Unsuccessful Attempts at Extraction of Foreign Bodies from the Ear.—LEUTERT (*Archiv für Ohrenheilkunde*, vol. lxi., Nos. 1 and 2) draws attention to the dangers attending the attempts at extraction of foreign bodies impacted in the external auditory canal when these efforts are made under the conditions of imperfect knowledge of the parts dealt with, the use of improper instruments, or the lack of proper illumination of the operative field.

A boy, aged four years, had, several days previously, thrust a pea into his left ear. The physician who was called attempted to remove the foreign body by means of a hair-pin and without the aid of proper illumination, ceasing his attempt only after causing both considerable pain and a sufficient bleeding to obscure the field.

On examination the right ear was found to be normal both as to appearance and hearing power.

The lining of the left auditory canal was much swollen, and in the meatus were blood and a trace of thin purulent discharge.

On syringing with a normal salt solution the fluid ran freely from the left nostril, and as symptoms of pain and fever were wanting, the ear was lightly bandaged and the patient told to return at the end of eight days unless unfavorable symptoms made an earlier visit advisable.

Eight days later the swelling of the canal had so far diminished as to make an inspection of the drumhead possible, and there was found a large perforation, including almost the whole of the drumhead anterior

to the malleus, which was in normal position. The presence of the foreign body was not determined.

At the end of another week the further subsidence of the swelling in the canal revealed the perforation in its full extent and a rounded foreign body lying within the middle ear upon the tympanic floor. Attempts at removal by syringing failing, because the fluid, instead of returning escaped through the Eustachian tube and nose; the auricle was reflected forward and the pea easily extracted by means of a blunt hook. The healing of the post-auricular wound was effected without incident, but treatment of the suppuration of the middle ear had to be subsequently continued.

From this and similar experiences, coupled with a review of the literature of the subject, the writer draws the following conclusions:

1. In all early cases, seen at first hand, the primary attempt at extraction should be made with the syringe, and should the first attempt fail, this treatment should be successively repeated, unless it is found that, in consequence of a perforation of the drumhead and escape of the fluid through the Eustachian tube, this measure is negatived.

2. When it has been definitely determined that removal of the foreign body by means of syringing is impossible, instrumental manipulation may be resorted to, but only under conditions of good illumination and with instruments which may be passed beyond the foreign body, between it and the canal wall, without injury to the deeper seated parts. If instrumental manipulation is found to be ineffectual without danger to the soft parts, the auricle should be reflected forward and the body extracted.

3. In the event of previous attempts at extraction which have resulted in injury to and swelling of the canal walls, this swelling should be allowed to subside before further attempts at extraction are made. In the case of symptoms which demand immediate interference and extraction, either through the lumen of the canal or post-aurally, this should preferably be done under an anæsthetic.

Rupture of the Drumhead Caused by a Blow upon the Ear.—MENIERE (*Archives Internationales*, vols. xvii., No. 3) relates a case of a young man, aged twenty-five years, who received a blow upon the left ear in boxing, the hand of his adversary being covered with the ordinary soft leather boxing-glove. The blow was followed immediately by a sensation of sharp pain in the depth of the ear and by slight vertigo. Examination of the ear, one hour later, showed a rupture of the drumhead about 4 mm. in diameter in the anterior-superior quadrant, partly concealed by a few drops of blood. The subjective symptoms were of a very moderate character, but little pain and a slight tinnitus.

The treatment consisted in the application of a warm douche of weak boric acid solution for three or four minutes and the subsequent stopping of the canal with a firm pledget of absorbent cotton; furthermore, the patient was advised, in the event of a desire to blow the nose, to abstain from the use of a handkerchief and to clear the nose by blowing gently with only one nostril closed at a time. In addition, the patient was directed to insufflate a warm boric acid solution.

Three days later the opening in the drumhead was closed by a clot, and on the eighth day after the accident cicatrization was complete and the hearing absolutely normal; there remained only the tinnitus as a

reminder of the injury, and this symptom also disappeared entirely at the end of fifteen days.

Traumatic ruptures of the drumhead, except such as are the result of direct violence, are most common among artillerymen and others following occupations which expose them to loud concussions. Cases of concussion rupture from a direct blow upon the outer ear are much less common, but the writer cites a similar case in which a slight buffet upon the auricle resulted in a rupture of the drumhead extending over three-quarters of its surface; treatment of the same character as that above given was employed, but the case was much more slow in healing, it being at least a month after the accident before this result was attained.

As the result of his experience in similar cases, the writer advises for treatment: 1. Disinfection of the parts by the use of peroxide of hydrogen. 2. Firm stoppage of the canal. 3. Avoidance of inflation of the middle ear.

OPHTHALMOLOGY.

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Ligation of the Lacrymal Canal to Prevent Infection following Cataract Extraction.—QUACKENBOS (*Boston Medical and Surgical Journal*, March 31, 1904) reports two cases of ligation of the lacrymal canal to prevent infection following cataract extraction. The usual preparations for cataract extraction were carried out. Cocaine was used, then the contents of the mucocele were squeezed out and the eye washed with corrosive, 1:5000. A small probe was inserted into the upper punctum and a suture passed around the canal from the inside of the lid out. This was repeated on the lower lid. The sutures were passed 2 mm. or 3 mm. from the punctum and were tied quite firmly. The usual operation for cataract was done and the healing was without incident. The stitches were removed at the end of two weeks in the first case, and nine days in the second case. The author believes that the stitches should be removed as soon as the corneal wound has healed firmly. He claims that the advantages of this method are "its simplicity, the ease with which it can be done, its freedom from pain, that it can be done at the time of the operation, and that it can be used even in doubtful cases."

The Epidemic of So-called Trachoma.—ALGER (*New York Medical Journal* and *Philadelphia Medical Journal*, April 9, 1904) makes some appropriate observations upon the scare on the subject of trachoma from which the people of New York, and, to some extent, of the whole country, have been victims. Beginning with the ruling of the United States immigration authorities, anxious to find some excuse for prohib-

iting the entrance into the country of so-called undesirable classes of immigrants, it has spread to the schools, from which thousands of children have been excluded because of their supposed infection with a dangerous contagious disease. Many of those who fomented the agitation were not altogether altruistic; some were influenced by race prejudice; others were eager for increased appropriations for their dispensaries.

Alger insists upon the great importance of distinguishing follicular conjunctivitis from trachoma. The former is a trivial matter. True trachoma is a real scourge leading to unending discomfort and suffering and often resulting in the most serious consequences to the sight. There can be no doubt that the vast majority of the thousands of children who were affected had simply the mild disease. The heroic measures with which many of them were treated were both cruel and useless. These views are not new. The best authorities fully endorse them, but they should be borne in mind and acted upon by ophthalmic surgeons who have to give an opinion when cases of suspected trachoma are brought to their notice.

Ethyl Chloride as a General Anæsthetic in Eye Work.—STEPHENSON and CHALDECOTT, London (*The Ophthalmoscope*, April, 1904) highly recommends this agent as being especially useful in eye work. It has several distinct advantages over all the older anæsthetics. It is easy to give. Anæsthesia is quickly reduced and its effects pass away rapidly. Vomiting is not common. Cyanosis or jactitation do not occur. A single dose is administered, which will certainly within a few seconds induce the necessary anæsthesia, but which is not sufficient to kill. If the operation is more protracted it is easy to administer more ethyl chloride or to continue the anæsthesia by means of chloroform. It is especially useful in permitting thorough examination in young children with intense photophobia and in the exquisitely sensitive corneas of patients suffering from gonorrhœal ophthalmia. Under its influence lacrymal probes can be passed, chalazia extirpated, canthoplasty performed, trachomatous granulations expressed, the anterior chamber tapped, corneal ulcers cauterized, lens and secondary cataracts needled, and the recti divided, etc.

Cases of Hydatid Cyst of the Orbit.—MARSHALL, of London (*The Ophthalmoscope*, April, 1904), reports an instance of this rare disease. The patient was a girl aged five years. The right eye had turned for about eighteen months; between the ball and the inferior and outer margin of the orbit a tumor the size of a muscatel grape, apparently fluctuating, was situated. The movements of the globe were free, although less than normal, especially restricted outward and downward. The eye appeared to be healthy and the pupil active; vision doubtful. The growth was removed in its sac. The cyst was found to be one-and-a-half inches in diameter, with all the characteristics of an hydatid cyst. There were no echinococcus heads in it, as is usually the case, for heads and hooklets are noted in only a small minority of cases of human echinococci. These cases are exceedingly rare. Prior to 1895 only 45 cases had been published. Since then others have been reported. There is one other situation, however, in which it is still less common to find the cyst, and that is in the eyeball itself. Marshall knows of only 3 cases of this condition.

PATHOLOGY AND BACTERIOLOGY.

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Is Human Tuberculosis and Tuberculosis of Domestic Animals Caused by the Same Variety of Micro-organism, Namely, the Bacilli Koch? (Internationaler Kongress für Hygiene in Brüssel, 1903.)—The results of the discussion upon this subject as given by LIGUIERES (*Cent. f. Bakt. u. Parasitenkunde*, 1904, Bd. xxxiv. p. 427) are that from the tuberculous lesions of man and animals specific bacilli can be isolated, which, though belonging to the same group of micro-organisms, yet possess sufficiently different characteristics to stamp them as distinct varieties. The bacillus of avian tuberculosis differs from that of human tuberculosis, both as regards cultural properties and pathogenicity. The bacillus of human tuberculosis differs from that of bovine tuberculosis in various and constant pathogenic criteria. Whereas the bovine bacillus produces a more or less general tuberculosis when injected into cattle, the bacillus of human tuberculosis when inoculated in the same manner calls forth only a local transient reaction, which heals and leaves no tuberculous infection demonstrable through lesions, presence of bacilli, or tuberculin reaction. In general, one finds the bacillus of avian tuberculosis in birds, the bacillus of human tuberculosis in man, and the bacillus of bovine tuberculosis in cattle, although the latter variety may exceptionally be demonstrated in man, especially in cases of primary intestinal tuberculosis. When the cases of accidental infection of man are considered, either through wounds or through ingestion of virulent milk from a cow which is the subject of tuberculous mastitis, the proof of the possibility of infection of man by means of the bovine tubercle bacillus is at hand. For this reason, it would be a great mistake to abandon the well-organized fight against bovine tuberculosis. This is of special importance, since tuberculosis is constantly increasing among cows.

On the Action of Hæmolysins in the Body.—FUKUHARA (*Ziegler's Beiträge*, 1904, Bd. xxxv. p. 434) studied the effects of intraperitoneal, subcutaneous, and intravenous injections of heterohæmolysins in rabbits, guinea-pigs, rats, pigeons, and chickens. These injections were frequently followed by a slight elevation in temperature, hæmoglobinuria, and evidences of marked renal disturbances. In one guinea-pig there was a bloody diarrhœa. Besides the solution of the hæmoglobin of the red blood corpuscles, other evidences of degeneration and signs of regeneration rapidly made their appearance in the circulating blood. Icterus was occasionally noted. Large doses of the hæmolytic sera were quickly fatal. Death was referred to the toxic action of the sera

upon various kinds of cell protoplasm. In the less acute or chronic cases the internal organs showed coagulation of the blood with plugging of the capillaries and small vessels by thrombus masses, hemorrhages noticeable particularly in the heart muscle and kidney, ulcers in the stomach and intestines, hæmoglobinæmia, and pigmentation of various organs. It was thought that the hemorrhages in the heart and cortex of the kidney were due to a thermolabile toxin of the hæmolytic sera, since they did not occur if heated sera were used. Small foci of necrosis were frequently found in the liver. In a severe case degeneration of the bone-marrow was found, while with milder intoxications proliferative changes of the bone-marrow cells were noted. The erythrocytes were particularly affected; the eosinophiles appeared very resistant. The injection into an animal of foreign erythrocytes and subsequent intravascular solution of these cells called forth a series of changes much like those following injections of heterohæmolysins. Bile may have the same destructive effect as hæmolysins upon the blood. The author concludes that the action of a cytolytic serum is not specific. Hæmolytic serum injures not only red blood corpuscles, but heart muscle, liver cells, and kidney cells.

Demonstration of the Bactericidal Reaction in the Blood Serum of Typhoid Fever Patients.—STERN and KORTE (*Berl. klin. Wochen.*, 1904, No. 9, p. 213), by using a method devised by Neisser and Wechsberg, have measured the bactericidal power of the blood serum of typhoid fever patients and of normal individuals. To fixed quantities of fresh unheated rabbit serum and a dilution of a twenty-four-hour bouillon culture of typhoid bacilli varying amounts of heated typhoid serum were added. After the mixture had remained at 36.5° C. for three hours the whole was plated. The authors find that almost complete bacteriolysis takes place, even when typhoid serum is used in very high dilutions. In a few cases the serum, when diluted 50,000 times, is still active. Sera from persons who have not had typhoid fever possesses a much lower bactericidal value, and usually is only effective in dilutions of 1 : 200 or thereabouts. No relationship can be made out, either between the severity of the disease or the tendency to relapse and the bactericidal value of the serum. Neither does there appear to be any direct connection between the agglutinating power of the serum and its bactericidal action. Sera diluted beyond the agglutinating point are still bactericidal. The authors suggest that this method of determining the bactericidal value of the blood serum may be of clinical value in the diagnosis of typhoid fever, especially when the Widal reaction is delayed in its appearance.

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CONTENTS.

ORIGINAL COMMUNICATIONS.

PAGE

"Two Thousand Operations for Appendicitis," with Deductions from His Personal Experience. By JOHN B. MURPHY, A.M., M.D. . 187

Pseudomelanosis of the Hæmolymph Glands. By ALDRED SCOTT WARTHIN, PH.D., M.D. 211

A Study of the Tubercle Bacilli Isolated from Three Cases of Tuberculosis of the Mesenteric Lymph Nodes. By THEOBALD SMITH, M.D. 216

A Study of Circumscribed and Diffuse Pulsation of the Wall of the Thorax. With the Report of a Case of Pulsating Hæmothorax. By JOSEPH SAILER, M.D. 225

Pyopericardium and Commencing Interstitial Pneumonia Complicating Ileocolitis in an Infant Aged Four and a Half Months. By D. J. M. MILLER, M.D., and J. C. GITTINGS, M.D. 248

The Treatment of Chronic Internal Hydrocephalus by Autodrainage. By ALFRED S. TAYLOR, M.D. 255

A Case of Splenomegaly with Blood Changes and Symptoms Resembling Those of Banti's Disease, Apparently Due to Malaria. By SOLOMON SOLIS COHEN, M.D., and RANDLE C. ROSENBERGER, M.D. . . 271

The Leukocytosis of Pregnancy, of the Puerperium, and of Eclampsia. By RALPH WALDO LOBENSTINE, M.D. 281

The Relation of the Islands of Langerhans to Diseases of the Liver, with Special Reference to Carbohydrate Metabolism. By JOSEPH C. OHLMACHER, M.D. 287

Orthostatic Albuminuria. By CHARLES LOUIS MIX, A.M., M.D. . . 307

Adiposis Dolorosa, with Report of Three Cases. By J. N. HALL, M.D., and C. E. WALBRACH, M.D. 318

Aneurysm of the Heart. By PHILIP D. BOURLAND, B.S., M.D. . . 323

Œsophageal Neuroses. By A. L. BENEDICT, A.M., M.D. 327

REVIEWS.

Progressive Medicine. A Quarterly Digest of Advances, Discoveries, and Improvements in the Medical and Surgical Sciences. Edited by Hobart Amory Hare, M.D.; assisted by H. R. M. Landis, M.D. . 333

American Text-book of Surgery for Practitioners and Students. Edited by William W. Keen, M.D., LL.D., F.R.C.S. (Hon.), and J. William White, M.D., Ph.D. 334

The Treatment of Neurasthenia. By A. Proust and Gilbert Ballet. 334

A Manual of Medicine. Edited by W. H. Allchin 335

Les Nerfes du Cœur Chez les Tabetiques (Etude Clinique et Anatomo-pathologique). Par le Dr. Jean Heitz 336

The American Year-book of Medicine and Surgery for 1904. Edited by George M. Gould, A.M., M.D. 337

The Lymphatics. By Delamere, Poirier, and Cuneo 337

A Manual of Fever Nursing. By Reynold Webb Wilcox, M.A., M.D., LL.D. 338

International Clinics. Edited by A. O. J. Kelly, A.M., M.D. . . 339

PAGE

Lehrbuch der Gynäkologie. By Max Runge 340
Blood Pressure in Surgery. By George W. Crile, A.M., M.D. . . 341
Chart of the Sympathetic Nerve (Life Size). By Byron Robinson . 342
Modern Microscopy. A Handbook for Beginners and Students. By M. I. Cross and Martin J. Cole 342
The Structure of the Glands of Brunner. By Robert Russell Bensley . 343
Charles White, F.R.S., A Great Provincial Surgeon and Obstetrician of the Eighteenth Century. By Charles J. Cullingworth, M.D., F.R.C.P., London 344
Nervous and Mental Diseases. By Archibald Church, M.D. and Frederick Peterson, M.D. 344
A Text-book of Operative Surgery, Covering the Surgical Anatomy and Operative Technic Involved in the Operations of General Surgery. By Warren Stone Bickham, Phar.M., M.D. 345
Diseases of the Skin: An Outline of the Principles and Practice of Dermatology. By Malcolm Morris 346

PROGRESS OF MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

WILLIAM OSLER, M.D.,

AND

W. S. THAYER, M.D.

The Diagnosis of Gastric Carcinoma 347
Factors Influencing the Coagulation of the Blood in the Living Organism 348
A Contribution to the Study of Paroxysmal Hæmoglobinuria . . 349
Pneumococcus Peritonitis 350

SURGERY.

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D.,

AND

F. D. PATTERSON, M.D.

The Surgical Treatment of Epilepsy 351

THERAPEUTICS.

UNDER THE CHARGE OF

REYNOLD WEBB WILCOX, M.D., LL.D.

Adrenalin in Ocular Therapeutics 354
Adrenalin in Glaucoma 354
Adrenalin in Cancers of the Respiratory Passages 355

PAGE
Iodine Glycerole in Granular Conjunctivitis 355
Intravenous Injections of Sodium Salicylate in Rheumatism . . 355
Quinic Acid in Gouty Conditions 355
Sodium Sulphanilate in Exophthalmic Goitre 356
The Treatment of Cardiac Insomnia 356
The Medical Treatment of Pyloric Stenosis 356
Isopral, a New Hypnotic 357
Intratracheal Injections of Eucalyptol in Pulmonary Tuberculosis and Chronic Laryngitis 357
Beer Yeast in Exanthemata and Erysipelas 357

PEDIATRICS.

UNDER THE CHARGE OF

LOUIS STARR, M.D.,

AND

THOMPSON S. WESTCOTT, M.D.

The Association of Chorea with Tænia Solium 358
Enuresis in Children 359
Influence of the Ingestion of Chloride of Sodium upon the Weight of Nurslings 359
An Early Case of Friedreich's Ataxia 360
Herpes Zoster Associated with Generalized Bullous Eruption from Prolonged Administration of Arsenic 360
A Contribution to the Study of Cerebrospinal Meningitis 361

OBSTETRICS.

UNDER THE CHARGE OF

EDWARD P. DAVIS, A.M., M.D.

Eclampsia, its Causation and Treatment 362
The Scar of Uterine Rupture Found in a Uterus Removed by Operation 364

GYNECOLOGY.

UNDER THE CHARGE OF

HENRY C. COE, M.D.,

ASSISTED BY

WILLIAM E. STUDDIFORD, M.D.

Vaginal Hysterectomy for Prolapsus 365
Precocious Menstruation 365
Treatment of Rupture of the Uterus 365

PAGE

Anatomy of Chronic Metritis 365
Gonorrhœal Endometritis 366
Primary Cancer of the Tube 366
Treatment of Retroversion 366
Gonorrhœa of Skene's Glands 366
Rectal Alimentation 367
Method of Shortening the Round Ligament 367
Treatment of Postoperative Intestinal Paralysis 367
Combined Cancer and Tuberculosis of the Uterus 367
The Curability of Cancer 367
Ureteral Surgery 368
Advantages of Abdominovaginal Drainage 368
Hæmatoma of the Ovary 368
Pneumococcus Peritonitis 368
Sarcomatous Degeneration of Fibroids 368

DERMATOLOGY.

UNDER THE CHARGE OF

LOUIS A. DUHRING, M.D.,

AND

MILTON B. HARTZELL, M.D.

A Papuloexudative Dermatosis Produced by Pilocarpine . . . 369
The Cutaneous Manifestations of Appendicitis 369
Lupus Erythematosus with Bullous Beginning 370
A Case of Larva Migrans 370
Paget's Disease 371
Tuberculosis of the Skin 371

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

SIMON FLEXNER, M.D.,

ASSISTED BY

WARFIELD T. LONGCOPE, M.D.

The Elastic Tissue of the Ventricles of the Heart 372
Improved Technique of Agglutination Test in Typhoid Fever; the Use of Formalized Cultures 372
The Chemistry of the Urine in Diseases of the Pancreas . . . 373
Change in the Spleen in Pernicious Anæmia 374